Journal

OF THE

AMERICAN VETERINARY MEDICAL ASSOCIATION

Cattle Abortion Due to Listeria

TWO REPORTS, one on bovine abortion due to natural infection, the other on experimental infection, with Listeria monocytogenes. Page 221

Communicable Disease Prevention

A CONSIDERATION of eradication versus control of communicable diseases by the director emeritus of the Pan American Sanitary Bureau. Page 234

Tranquilizers for Weaning and Shipping Calves

AN EVALUATION of the effect of tranquilizers on calves being weaned and on those being shipped. Page 240

ALSO see editorial on tranquilizers. Page 267

Hemophilia B in Dogs

A STUDY of a condition in dogs resembling Christmas disease in man. Page 247

Vol. 137 August 15, 1960 No. 4

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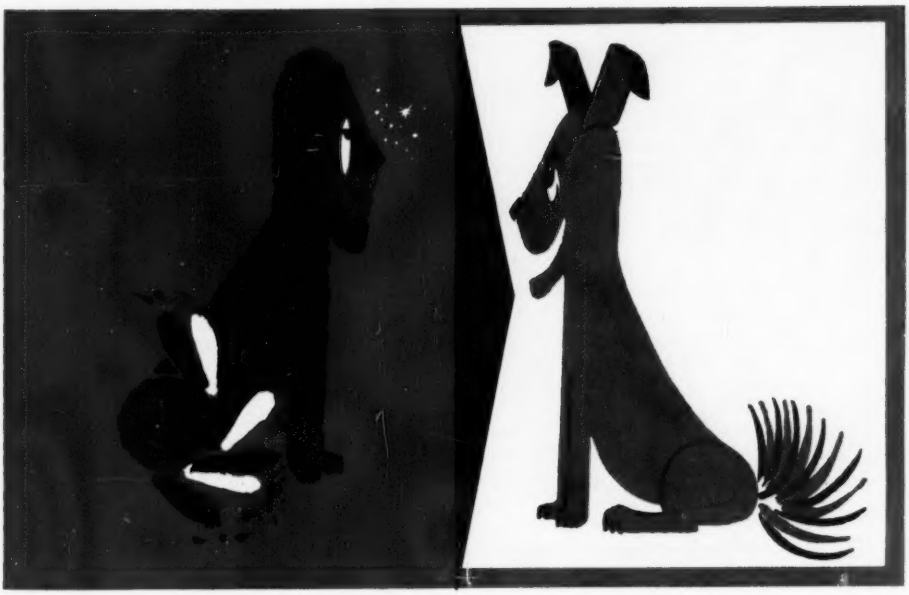

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Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION

Vol. 137 No. 4 Aug. 15, 1960

CONTENTS

Contents continued on adv. page 4

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Correspondence

Brucella Vaccine for Fistulous Withers

June 9, 1960

Dear Sir:

I read with interest the short note on fistulous withers (JOURNAL, June 1, 1960, page 549).

In 1943, the JOURNAL reported work of 2 Connecticut men in this connection. Since that time, I have personally treated 40 to 50 horses and mules that had fistulous withers and several that had poll-evil. Of the number treated, I tested more than 50 per cent for brucellosis and found, without exception, a titer. These animals were given Brucella vaccine over a period of 6 to 8 weeks. The inoculations were spaced 3 weeks apart, the number of inoculations being dependent upon the subsequent degree of improvement of the fistula. The majority of these fistulas, perhaps 90 per cent, completely cleared up, and there were few recurrences. Some animals required supportive treatment, such as sulfanilamide and penicillin.

The majority of animals with fistulas were cow ponies on ranches that were heavily contaminated with Brucella organisms.

It is interesting (per an article some months ago in the JOURNAL) that blood serums from naturally infected animals give an agglutination reaction with irregular flocculation, but that serums from those carrying titers from vaccination with strain 19 have a uniform flocculation. These facts have been known for nearly 20 years.

s/F. W. GROHE, D.V.M.
8945 Katy Road
Houston, Texas

Tritrichomonas or Trichomonas?

June 15, 1960

Dear Sir:

We are confused because of the inconsistent use of terms to describe *Tritrichomonas foetus* in the May 15 issue of the *J.A.V.M.A.* In the beginning of the article, Tritrichomonas is used. In the summary, Trichomonas is used. Which is correct?

s/RICHARD D. BURNS, *D.V.M.*
Senior Veterinary Editor
Scientific Information Division
Eaton Laboratories
Norwich, N. Y.

[Editor's Note: The author of the article, Dr. B. O. Brodie of the University of Illinois, chose to use Tritrichomonas in his original manuscript, and it was an editorial oversight that Trichomonas was used in the summary of the published article.

Although the AVMA has used *Trichomonas foetus* in articles in past years, Tritrichomonas is not incorrect. It is the preferred generic term of R. R. Kudo, professor of zoology at the University of Illinois and author of *Protozoology* (Charles C Thomas, 1954), and of R. P. Hall of New York University, author of *Protozoology* (Prentice-Hall, 1953). Judging by Kudo's work, it seems that the organism was originally named *Tritrichomonas foetus* by an early investigator, C. A. Kofoid.]

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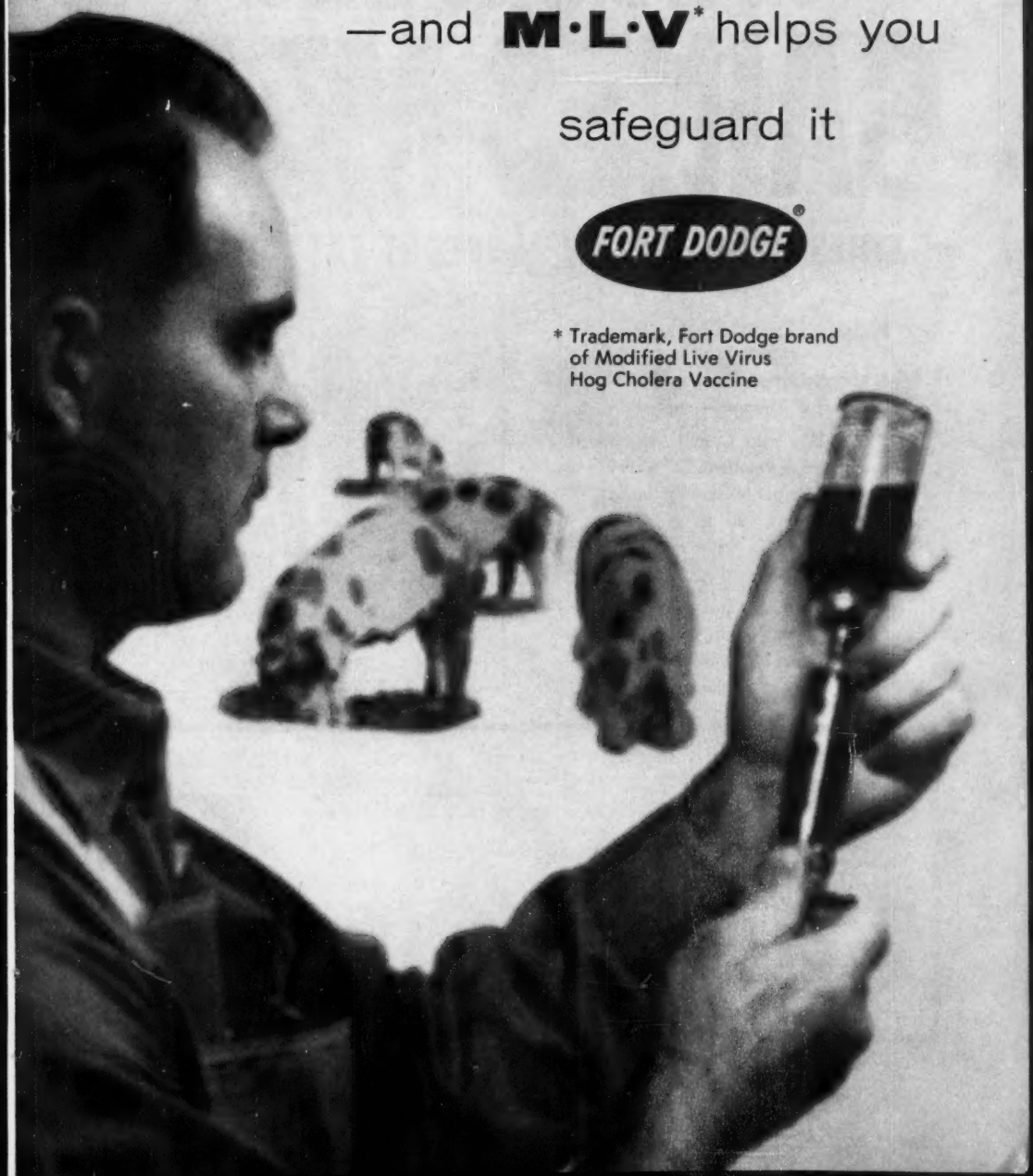

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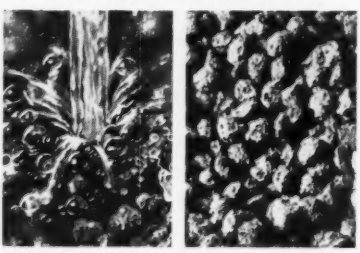

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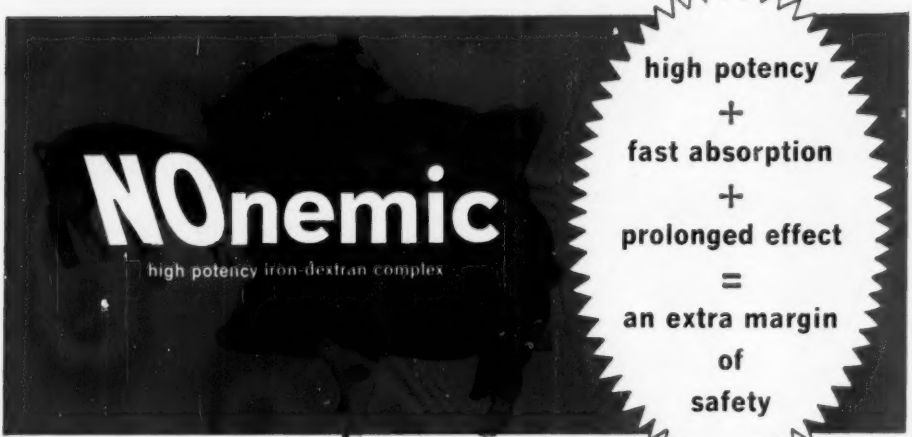

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RESULTS: The following remarks are abstracted from actual clinical investigation reports on the use of SULFABROM SOLUTION on file at the Merck Animal Science Research Department. (1) "*Symptoms:* Recently shipped-in cow. Temperature 106°. Labored breathing. Rales audible over chest area. *Diagnosis:* Pneumonia (shipping fever complex). *Treatment:* 700 cc. SULFABROM SOLUTION in one dose daily for two days. Sixty grams of SULFABROM BOLUSES given at time of I. V. injection. *Results:* Temperature returned to normal in 24 hours". (2) "*Symptoms:* Dystocia with removal by forced extraction of putrified fetus. Temperature 104°. Rapid pulse and breathing. Anorexia. *Diagnosis:* Metritis. *Treatment:* Two doses SULFABROM SOLUTION 700 cc. each. *Results*: Temperature 101.6° in 24 hours. Appetite returning". (3) "*Symptoms:* Foul hoof—limited to 'between the toes' variety with swelling. *Diagnosis:* Foot rot. *Treatment:* 700 cc. SULFABROM SOLUTION *Results:* Eight of 11 animals walked normally in 48 hours with swelling reduced to normal or near normal. Three animals required second treatment and returned to normal in 48 hours."

These quoted reports are typical of those being received from clinical investigators and from veterinarians like yourself. We believe they demonstrate that *the only sulfas available to you exclusively*—new SULFABROM SOLUTION and SULFABROM BOLUSES—are also the most effective you can use.

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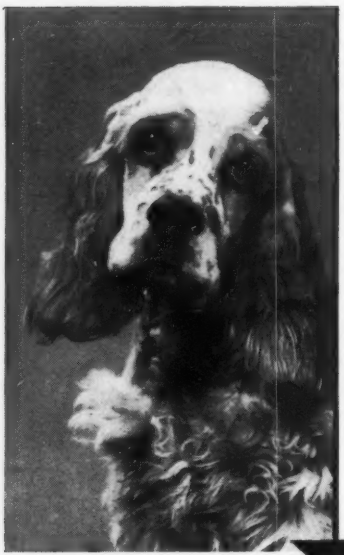

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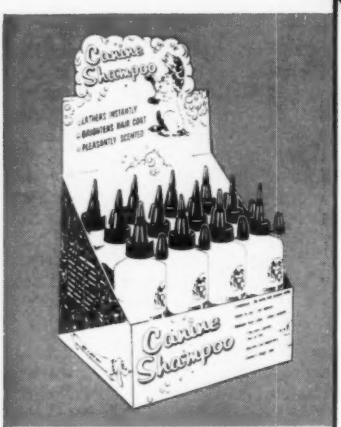

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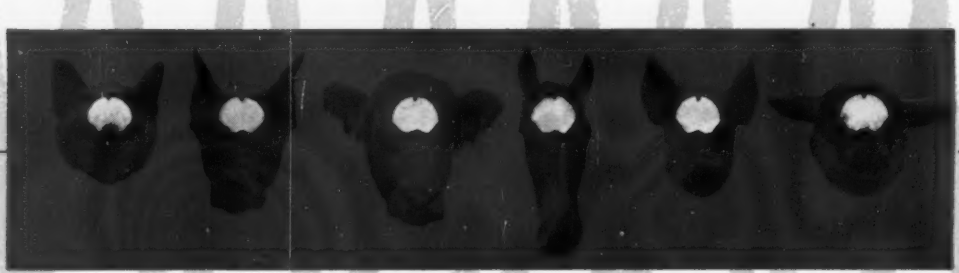

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news from Washington . . .

FROM THE AVMA WASHINGTON OFFICE
J. A. McCallam, VMD
Brig. Gen. USA (Ret.)

The following have become public laws:

USDA appropriations for fiscal year 1961: *P. L. 86-532, H. R. 12117.* Also *P. L. 86-651, H. R. 12740.*
Amendment of Food, Drug, Cosmetic Act: *P. L. 86-537, H. R. 7480.*
Amendment to Humane Slaughter Act of 1958: *P. L. 86-547, H. R. 12705.*
International Health and Research Act: *P. L. 86-610, S. J. Res. 41.*
Color additives amendment of 1960: *P. L. 86-618, S. 2197.*
Support of land grant college instruction: *P. L. 86-658, S. 3450.*
Donation of surplus property: *P. L. 86-570, S. 1018.*
Postal rate revisions: *P. L. 86-644, H. R. 4595.*

NEW BILLS

Require Generic Name in Drug Advertisements

H. R. 12831, Rep. Dingell (D., Mich.), to amend Federal Trade Commission Act to require drug advertisements to contain certain information. In effect proposes that a chemical compound dispensed on prescription as a drug under trademark, brand name, etc., shall be misleading unless it includes the generic name of the chemical compound.

Strengthen Food, Drug, Cosmetic Act

S. 3815, H. R. 12949, identical bills, introduced by Sen. Hill (D., Ala.) and Rep. Harris (D., Ark.), respectively, would amend Food, Drug, and Cosmetic Act to strengthen existing inspection authority; require manufacturers of new drugs to keep records and make reports based on clinical experience, etc., bearing on permissibility of such drugs; require adequate controls to insure quality, and extend to all antibiotics the certification provisions now limited to certain antibiotics.

Authorize Federal Loans for College Facilities

S. 3776, H. R. 12930, H. R. 12933, identical bills by Sen. Clark (D., Pa.), Rep. Metcalf (D., Mont.), Rep. Thompson (D., N.J.), respectively, would authorize federal loans and matching grants as alternative forms of assistance to colleges and universities for construction, alteration, conversion, etc., of classroom buildings and other academic facilities.

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WASHINGTON NEWS—Continued

Amend Food and Drug Act

S. 3677, Sen. Kefauver (D. Tenn). To amend Food and Drug Act to redefine term "new drug," provide for licensing of persons engaged in propagation, manufacture, or preparation of drugs dispensed only upon prescription.

Increase Social Security Benefits

S. 3725, Sen. Saltonstall (R., Mass.). To amend Social Security Act and Internal Revenue Code. Would increase minimum benefits, earnings on which benefits based ($4,800 to $6,000), and amounts of earnings permitted without loss of benefits. ($1,200 to $2,400). *Note:* The Senator intends this Bill be considered along with proposal passed by House.

Treatment of Experimental Animals

H. R. 12757, Rep. Oliver (D., Maine). To provide for humane treatment of animals used in experiments and tests.

MISCELLANEOUS

ARS Amends "Diseased Specimens" Regulations

Agricultural Research Service, USDA, amends Meat Inspection Regulations regarding diseased specimens released for educational, research, and other nonfood purposes, *Federal Register* June 28, 1960. Written application to inspector in charge on M. I. Form 403-10 required for permit to obtain specimens. Permit good for one year; may be revoked under certain conditions. Item stated the amendment simplifies procedure for obtaining release from inspected establishments of specimens for research and other nonfood purposes, now eligible for such release.

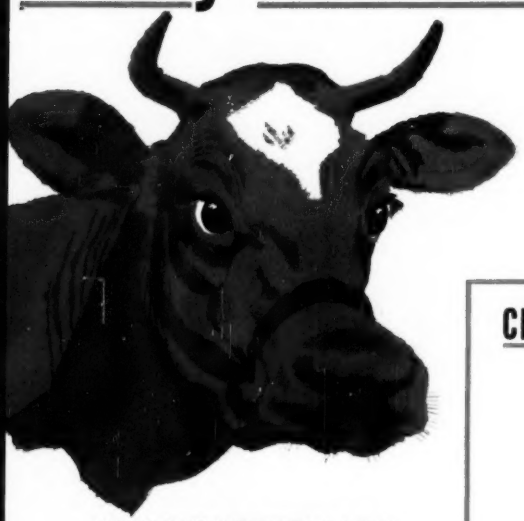
USDA Issues Carcass Identification Policy

A statement of policy regarding identification of carcasses of animals slaughtered and handled under the Humane Slaughter Act of 1958 (*P.L. 85-764*) issued by USDA, and published in *Federal Register* June 25, 1960. Periodically the Director Meat Inspection Division, ARS, will publish in the *Federal Register* a table listing the official establishments which use humane methods and handling, the establishment numbers, and the species of livestock being slaughtered in accordance with such methods. Additions and deletions will also be published.

Courses Open to VC Reserve Officers

Courses available to U.S. Army Veterinary Corps Reserve Officers of approximately 14 days active duty training at Brooke Army Medical Center, Fort Sam Houston, Texas.
Course 8-A-C11, AMedS *Field Grade Officer* Refresher, Oct. 9 to 22, 1960, to Jan. 29 to Feb. 11, 1961.
Course 8-A10, AMedS *Company Grade Officer* Refresher, Oct. 9 to 22, 1960, and Jan. 29 to Feb. 11, 1961. Submit request through Reserve channels to the appropriate Reserve Corps Headquarters.
Also 2 refresher courses, *8-A-F9,* will be held at the Army Meat and Dairy Hygiene School, Chicago, Ill.—one in February, the other in May, 1961, each 2 weeks. Additional information on these will appear in the Journal Sept. 1, 1960.

New URISED® Bolus for rapid relief of urinary infections in large animals



Relieves many urinary disorders—attacks both smooth muscle spasms and urinary tract infections!

Check these outstanding advantages:

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- **Attacks both gram-negative and gram-positive organisms all along the urinary tract**
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prevents milk fever relapse

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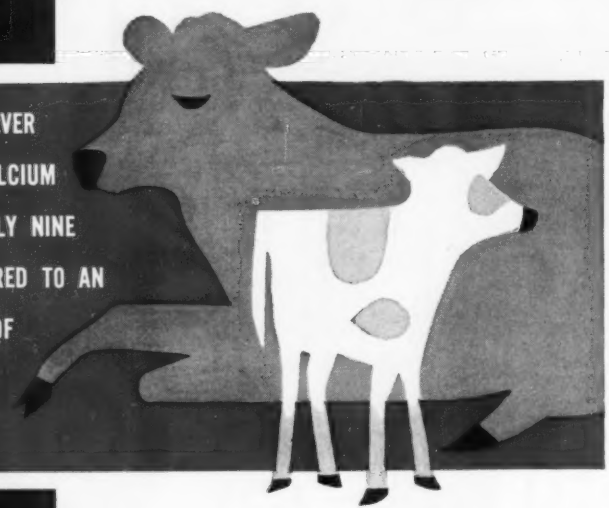
RECORDS OF 351 MILK FEVER CASES TREATED WITH CALCIUM AND PARTEROL SHOW ONLY NINE RELAPSES (2.5%), COMPARED TO AN AVERAGE RELAPSE RATE OF 22.8% WITH CALCIUM ALONE.[1,2,3]



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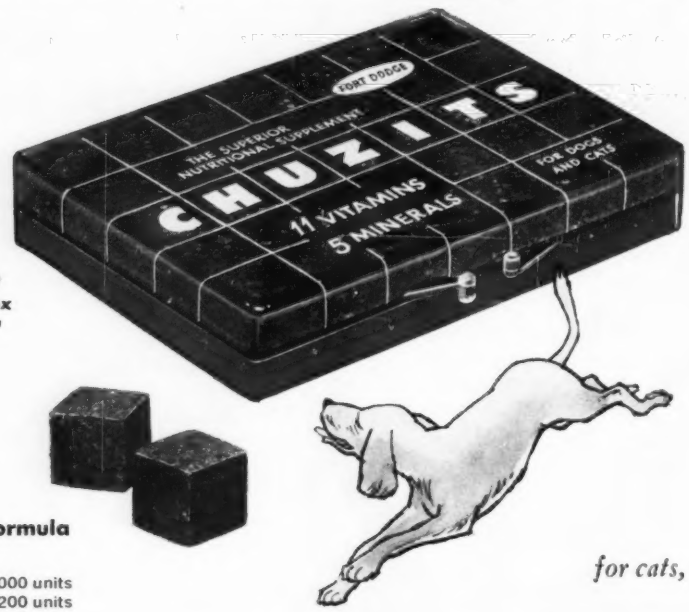

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Cattle Abortion

Associated with Natural Listeria monocytogenes Infections

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THE VETERINARY PROFESSION has made notable progress in determining the causes of abortion in cattle and developing appropriate means for the control of several diseases involved. Investigations continue to reveal the multiple causes that can be associated with premature expulsion of the fetus. When diseases such as brucellosis, vibriosis, trichomoniasis, and leptospirosis are eliminated, it is still found that important numbers of abortions occur. Of 188 fetuses examined during a 4-year period at the School of Veterinary Medicine (University of California), 71 per cent fell into no recognized diagnostic category.[6] Such figures point out the magnitude of the problem and the need for additional knowledge relating to the several causes which bring about the single effect of abortion. This paper deals with the role of *Listeria monocytogenes* in cattle abortion in California.

Field Investigations

Herd A.—A 400-cow commercial beef herd, composed principally of the Hereford breed, is in Siskiyou County at the northern end of the state. The problem there has been characterized by expulsion of the fetus late in gestation, frequently with systemic effects on the dam. Some of the abortions occur in the sixth and seventh months of gestation, but many occur in late pregnancy, with some calves born alive and dying soon thereafter. Weak calves can occasionally be nursed back to health and become normal animals. Late abortions are more apt to have severe effects on the dam, with visible depression, high temperature, frequent retention of fetal membranes, and the development of purulent genital exudates. Following intrauterine death, some calves become emphysematous or malpositioned. Abortions in the herd have occurred in successive years, with some cattle aborting more than once.

In 1959, the calving period was to start in mid-January. The ranch was visited on January 9, at which time 7 cows had aborted and 1 had died. Six cows were examined 12 hours to 4 days after they had aborted. Their body temperatures ranged from 103.9 to 105.5 F. Uterine discharges from all but 1 were yellowish, watery, fetid fluids. Bacteriologic examination of the 5 foul-smelling exudates did not reveal *L. monocytogenes*, though expected populations of coliforms, cocci, and other contaminating organisms were numerous. One cow (No. 8, table 1) had given birth to a live premature calf during the night preceding the visit. The cow had a tempera-

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ture of 104 F. and retained fetal membranes. The exudate was blood-tinged and mucoid but had no offensive odor. *Listeria monocytogenes* was isolated from this exudate. When the cow was examined, a placentome was removed from the uterus which also yielded the Listeria organism. The calf was not examined.

The uterus and vagina were available from a cow that had died and been necropsied on the previous day. The organs showed marked lesions of metritis, with edema, fibrin on the serosal suraces, and hemorrhages on the mucosa. Exudate from the uterus and peritoneal fluid from the cow were cultured, but were heavily contaminated and yielded nothing of significance.

The fetus of cow 5, aborted 24 hours previously, was necropsied. The tissues were well preserved because the carcass had been chilled. The size of the calf, together with the eruption of its incisor teeth, suggested that gestation was nearly complete. The lungs had been aerated, indicating that the calf was alive at birth. A few petechiae and somewhat larger hemorrhages occurred on the surfaces of the heart and in the mucosa of the trachea. Blood-tinged straw-colored fluid was seen in the peritoneal cavity. Cultures were made from the peritoneal fluid, stomach contents, heart, liver, spleen, kidneys, and lungs. Tissues from the lungs, kidneys, spleen, and liver were free of the usual contaminating organisms encountered in field collections. *Listeria monocytogenes* was recovered from the fetal liver (table 1). The culture from the dam's genital tract yielded no Listeria organisms, even though the abortion had occurred only 1 day before collection of the genital exudate. At that time it had contained many contaminating bacteria.

Herd B.—Also in Siskiyou County, herd B was on land converted from dairying to beef cattle raising in 1953. Abortions occurred first in 1955. In 1956, 40 calves were lost from a herd of about 130 cows. In 1957, there were 10 abortions. Abortions occurred late in pregnancy in cattle of varying ages, with some systemic effects on the cows.

The breeding program had been arranged to have calving in 1959 begin in February. When the herd was visited in late January, 8 abortions had occurred. Uterine exudate was collected from 5 cows that had aborted 1 or 2 days before the visit. Two of the cows had retained fetal membranes. Fever reactions in the group ranged from 102 to 104 F. Ages varied from 3 to 9 years. Exudates from 4 of the cows were not cultured until 4 to 6 days after abortion. These samples yielded no organisms of significance. The sample from cow 5 was cultured 72 hours after abortion (collected 1 day after abortion, with 48 hours in transit to the laboratory). *Listeria monocytogenes* was obtained from this sample (table 1). The fetus of this cow was necropsied. The state of degeneration of the tissues suggested that the fetus had died *in utero* during the eighth month of gestation. The only gross changes were accumulations of red, watery

TABLE 1—Isolations of *Listeria monocytogenes* from Naturally Infected Cattle

Herd A			Herd B			Herd C		
Specimen	*Or.	**Re.	Specimen	Or.	Re.	Specimen	Or.	Re.
Uterine exudate cow #3	−	−	Uterine exudate cow #4	−	−	Uterine exudate cow #1	−	−
Uterine exudate cow #4	−	−	Uterine exudate cow #5	−	+	Uterine exudate cow #2	−	−
Uterine exudate cow #5	−	−	Pleural fluid from fetus of cow #5	−	+	Uterine exudate cow #3	−	−
Uterine exudate cow #6	−	−	Uterine exudate cow #8	−	−	Uterine exudate cow #4	−	−
Uterine exudate cow #7	−	−	Uterine exudate cow #9	−	−	Uterine exudate cow #5	−	+
Uterine exudate cow #8	−	+	Uterine exudate cow #10	−	−	Uterine exudate cow #6	−	−
Placentome cow #8	−	+				Pleural fluid fetus A	+	−
Uterine fluid dead cow	−	−				Liver — fetus A	+	+
Peritoneal fluid dead cow	−	−				Stomach contents fetus A	+	−
Liver from fetus of cow #5	−	+				Kidney — fetus A	+	+

*=Original culture; **=reculture; +=Listeria monocytogenes isolated; −=negative result.

fluids in the serous cavities and a uniform staining of the subcutaneous tissues. Also cultured 72 hours after abortion were organs from the fetus including liver, spleen, lungs, heart, stomach contents, and pleural fluid. Several of the tissues were contaminated, but the pleural fluid yielded *L. monocytogenes*. Thus the organism was obtained from both dam and fetus.

Herd C.—This herd was composed of cross-bred Hereford and Angus cattle, in Monterey County in the central coastal area of the state. It consisted of approximately 300 females of breeding age. For 3 successive years, abortions had occurred in 3- to 7-year-old cattle. In 1956, the calf crop was 50 per cent, with 17 known abortions; in 1957, there were 11 abortions. When the ranch was visited, on Oct. 9, 1958, 15 calves had been aborted. The first abortion was on August 21; the herd had been bred to begin calving in October. There were 12 stillbirths and 3 calves that died shortly after birth. Retention of fetal membranes occurred in some cows, in addition to systemic effects such as fever and lassitude.

Six cows which had recently aborted were examined. Their temperatures ranged from 102 to 105 F. Hyperemia was noted in the vagina, with variable accumulations of mucus and pus on the vaginal floor. One cow had retained fetal membranes in an advanced stage of decomposition. Four of the cows had presumably aborted several days previously since their uterine horns had involuted to a diameter of 1 to 1½ inches. Exudates from the anterior vagina were cultured. *Listeria monocytogenes* was obtained from the specimen from cow 5 (table 1).

The herd was again visited in April, 1959, and semen was collected from 5 bulls. On culture, the semen yielded only organisms common to the prepuce and skin. In September of 1959, the time of this writing, 3 more abortions occurred. One fetus was available for study. There were no gross lesions, but *L. monocytogenes* was isolated from the liver, kidneys, pleural fluid, and stomach contents (table 1). The annually recurring nature of the disease was evident by this event.

Bacteriologic Studies

Exudates and tissues were subjected to bacteriologic examinations for recognized pathogens among the order Eubacteriales, Among the substrates were tubed thiol medium and bovine blood agar plates incubated at 37 C. under increased CO_2 tension for cultivation of Vibrio and Brucella. Bovine blood agar plates and blood agar containing 0.05 per cent potassium tellurite were also incubated at 37 C., but under normal atmospheric conditions. Portions of the exudates and tissues were placed in tubes containing tryptose broth and held 6 weeks in the refrigerator (4 C.). They were then recultured on blood agar and blood agar containing tellurite. Reculture is always performed at this laboratory if Listeria is suspected, since a high percentage of infected tissues do not yield positive cultures on the original bacteriologic examination.[7] The phenomenon was well demonstrated in this study. Original culturing failed to yield Listeria in 6 of the 10 isolations (table 1).

The organism was isolated from 3 fetuses obtained from 2 herds. Eighteen uterine exudates were cultured. Most of the uterine exudates contained varying bacterial populations, since sufficient time had elapsed following abortion for organisms in the environment to contaminate the tissues. The agent was obtained from 1 exudate sample in each of the 3 herds.

Because most of the isolations were from specimens containing a mixture of bacteria, identification as *L. monocytogenes* required careful examination. All of the Listeria strains were motile gram-positive rods which produced a hemolysin that caused *beta*-type hemolysis of bovine erythrocytes. Uniform turbidity developed in broth cultures, and serum was not needed to support growth in broth. Broth cultures applied gently to the conjunctiva of guinea pigs produced typical keratoconjunctivitis. Subcutaneous injection of broth culture into mice caused death, with the expected lesions of focal necrosis in liver and spleen.

The strains were all agglutinable on spot tests with anti-Listeria serum. On physiologic tests, only minor variations in behavior were seen. Carbohydrates were fermented with the production of acid but not gas.

The tests were performed in a sterilized broth base to which 1 per cent of the test carbohydrate was added and then subjected to autoclaving for 15 minutes at 10-lb. pressure. Glucose, salicin, trehalose, and mannose were fermented promptly. Fermentation on lactose, maltose, rhamnose, starch, and dextrin was slight or delayed.

Sucrose, mannitol, dulcitol, sorbitol, raffinose, xylose, inulin, and glycerol were not attacked. Nitrates were not reduced. Growth in litmus milk produced slight acidity, and some reduction near the bottom of the tube.

Serologic Studies

The 6 Listeria isolates obtained in the fall and winter of 1958-1959, typed serologically, were found to belong to serotype 4b.*

Two serologic tests were applied to serums obtained from the involved herds. Somatic agglutination tests were run according to a procedure described previously.[8] The second test, called the antigen-fixation test, developed in our laboratory, has promise or detection of immune antibodies in serums containing the ubiquitous normal antibodies that also combine with Listeria antigens. The test employs chromatographic procedures and will be described elsewhere.

In herd A, serum was obtained from 19 cows, 13 of which had aborted. The agglutination titers varied from 1:100 to 1:800, with a geometric mean of 1:280. Blood samples were collected from 7 of the aborting animals 75 days after the first blood samples were collected. Six of the 7 cows had increases in titer on the second sampling, varying from 1 to 4 doubling dilutions. The geometric means for this double set of 7 samples changed from 1:82 to 1:490. Four positive antigen-fixation tests were obtained, all among the 13 aborting cows. Listeria was obtained from the fetus of a cow that had a threefold increase in agglutination titer but a negative antigen-fixation test. Uterine exudate of another cow, from which a blood sample was collected at the time of abortion, yielded *L. monocytogenes*. It had a 1:100 agglutination titer and a positive antigen-fixation test.

Twenty-one serum samples were obtained from herd B. Twenty of these, tested for somatic agglutinins, gave titers ranging from 1:200 to 1:800 with a geometric mean of 1:390. Antigen-fixation tests on 12 cows that had aborted revealed 6 positive reactions. Listeria was obtained from the uterine exudate of 1 cow with a 1:200 agglutinin titer and a positive antigen-fixation test. Five bulls, included in the testing, all gave positive antigen-fixation tests. One additional serum fixed the antigen from a group of 4 cows that had not aborted.

Serums were tested from 15 animals in herd C, 10 of which had aborted. The agglutination titers ranged from 1:200 to 1:800, with a geometric mean of 1:350. After 3 months, blood samples were collected from 5 cows that had aborted. Three of the 5 titers increased at the second sampling, and the geometric means increased from 1:300 to 1:460. Five serums were positive on the antigen-fixation test. Among these 5 were a cow from which the agent was isolated, 2 more aborting cows, and 2 cows that had not aborted.

Discussion

It has been known since 1939 that *L. monocytogenes* can produce abortion in cattle,[4] but invasion of the central nervous system is too often the only pathogenic manifestation anticipated from the agent. On the contrary, a review of world literature by Gray[5] indicates numerous instances where this bacterium has caused abortion, stillbirth, and neonatal death in man, cattle, sheep, and other animals. Recognition of the multiple pathogenic propensities of the agent is bringing it to the attention of public health investigators for more critical study.[12] This disease is another one of the zoonoses in which the veterinary profession should rightly play the key role in determining the relationship of the organism to disease in man as well as animals.

In California, the organism has been encountered sporadically from aborted ruminant fetuses at the School of Veterinary Medicine and in the California State Livestock and Poultry Pathology laboratories.[1] As in other states, these have been chance isolations made by routine diagnostic procedures. Studies made in Montana,[15] North Dakota,[2] and the work described here are among the few recorded herd studies in the United States made to investigate the role of *L. monocytogenes* in cattle abortions.

Question could arise as to the proper interpretation to place on these isolations

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of the Listeria organism. As far as is known, *L. monocytogenes* does not occur free in nature. It is always associated with the parasitic state, which means that its isolation from a tissue location, such as the genital tract, can be assumed to mean that it was living in or on the tissues, even though other organisms contaminate the sample. The pool of exudate in the anterior vagina had ample opportunity to become contaminated with various organisms from the external environment. This population of contaminating bacteria resulted in the development of odors and a greater accumulation of pus than existed at the time of abortion, as shown in cow 8 in herd A. The exudate there was a blood-tinged nonodorous, watery fluid that yielded *L. monocytogenes*.

The ease with which Listeria can be isolated from the genital tract following abortion is probably governed by 2 factors. First, the amount and kinds of competing microbial population that get into the sample can exert an effect on either destroying the pathogen or overgrowing it on culture. Second, the time elapsing between abortion and actual culturing is a factor. The period following abortion and collection of the sample is important since the genital tract appears to sterilize itself, eliminating the Listeria organism in a matter of days—just as it does with brucellosis and certain other specific genital infections.[9,13]

The time between collection and laboratory examination is also vital to success in isolation. Other critical factors are the care taken to avoid contamination when the sample is obtained and the conditions under which it is stored while in transit. Accurate information was available on the age of the specimens cultured from 11 uterine exudates, 3 fetuses, and 1 placentome. The 9 successul isolations from this group were made from cows which aborted 12 to 24 hours (av. 0.7 days) before specimen collection, with 1 to 3 days (av. 1.9 days) elapsing before the specimens were cultured. All specimens were kept chilled in either a refrigerator or ice chest. Bacteriologic results were negative from 9 uterine exudates involving abortions that occurred 1 to 4 days (av. 2.2 days) before specimen collection, with 2 to 4 days (av. 3.2 days) elapsing before they were cultured. The 1 specimen from which Listeria was obtained on original culturing was the fetus in herd C, aborted in September, 1959. This was the freshest specimen examined, with only 36 hours elapsing between abortion and culturing.

Certain aspects of differential diagnosis need to be considered. Specimens cultured for Brucella organisms from these herds were uniformly negative. Thirty-four serum samples from cattle that had aborted were all negative for Brucella agglutinins. The herds have participated in the brucellosis control programs and are considered Brucella-free. Herds A and C had been vaccinated twice against leptospirosis without any apparent change in the abortion problem. The clinical picture did not resemble Leptospira abortion, but some of the serums had reacted annually in the Leptospira agglutination test, which stimulated vaccination in the hope of eliminating the problem. Trichomoniasis was not considered a likely factor; the abortions were too late in gestation and the evidence of infertility was not as extensive as that encountered in trichomoniasis. Likewise, *Vibrio fetus* could not be demonstrated by cultural means.

Serologic study indicated that the *L. monocytogenes* strains affecting these herds were of the same serotype. This serotype, 4b, has been encountered frequently from various hosts and appears to be the most common type in California. The fact that Listeria abortion in cattle was caused by a serotype associated with listeriosis in other species permits the expectation that all of these Listeria infections can be interrelated.

The diagnostic use of serology in listeriosis is not yet precise enough for routine application. While the interpretation of serologic results on individual animals was sometimes difficult, certain conclusions could be drawn relating to the herd problem. There was evidence of rise in agglutinin titer in 9 of 12 paired serum samples, geometric mean agglutinin titers were higher than those expected in normal cattle, and 21 of 55 serums were positive on the antigen-fixation test. Experience with the antigen-fixation test is limited, but it has not given false positive results when tested with serums of several species. Tests performed on 67 normal cattle serums were uniformly negative on the antigen-fixation test. In summary, serologic evidence supported the conclusion

that Listeria infections were numerous in the 3 herds under study.

The fact that Listeria infection in these herds was not associated with recognized disturbances of the central nervous system is in accord with observations made elsewhere. There are several epizootics on record where genital infections occurred in the absence of encephalitis.[2,4,11,14] The clinician should also expect that the 2 syndromes may sometimes occur simultaneously in a given herd.[2,10] Listeria infections as they occurred here were associated with abortion in the last trimester of gestation, with fetuses killed *in utero* and then expelled, or born alive and usually dying in a short time. The effects on the dam were sufficiently severe to produce febrile responses, with enough metritis to cause frequent retention of the fetal membranes and, perhaps, occasional deaths. Death of the dam could not be considered a specific effect of the Listeria process since endometritis following abortion easily became complicated with several other organisms.

The bacteriologic studies again stress the necessity for using the reculture procedure to establish a diagnosis of listeriosis. Reculture is admittedly time-consuming and awkward. This fact should stimulate bacteriologists to study the mechanisms involved and look for reliable ways of establishing rapid diagnoses. Until that is done, diagnosticians will be obliged to use the more involved method of reculture, and to assume that they have not eliminated *L. monocytogenes* as the etiologic agent of either abortion or encephalitis until the results of reculture are available. At present, research workers have only scattered information on the incidence of genital infections with Listeria and its resultant role in public health and economic problems. This information can be added to only by diligent search for the agent in natural infections. The diagnostic experiences in this investigation may help guide others in their studies.

Summary

1) *Listeria monocytogenes* was isolated from 3 herds with a disease problem characterized by abortions in the last trimester of gestation; fever in the dam, with frequent retention of fetal membranes; and fetuses aborted either dead or alive.

2) The need to reculture specimens when searching for Listeria was demonstrated, since 6 of the 10 isolations were obtained only on reculture.

3) Serologic evidence suggested that there were numerous Listeria infections in the herds involved.

4) The recurring nature of the disease was observed in one herd where the Listeria organism was isolated during succeeding calving periods.

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Abortion of Cattle

Experimentally with Listeria monocytogenes

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LISTERIA MONOCYTOGENES has been cultured from aborted bovine fetuses in various parts of the world,[6] but studies with cattle on experimental reproduction of abortion are relatively few.[5,7] Isolation of the Listeria organism from bovine fetuses and the genital tracts of cows in 3 herds has been reported elsewhere.[9] Questions raised by that study were: Could the isolated strains of *L. monocytogenes* induce abortion, and what could be learned relative to the field problem by following the events in experimental infections? Accordingly, a representative strain from each of the 3 herds was used to infect pregnant heifers.

Materials and Methods

The cultures of *L. monocytogenes* used have been described in detail[9] and will be identified here as strain 5-59 (isolated from the uterine exudate of cow 8 in herd A), strain 4-59 (isolated from the uterine exudate of cow 5 in herd B), and strain 18-58 (isolated from the uterine exudate of cow 5 in herd C). Prior to cattle inoculation, the strains were injected into mice to preclude any loss of virulence that may have developed while they were on artificial mediums. Suspensions for inoculation were prepared by seeding flasks of brain-heart infusion broth directly from infected mouse organs. Flasks were incubated at 37 C. for 24 hours. When the bacteria was inoculated into cattle, pour plates were prepared to enumerate the suspensions by the plate-count method.

The animals were 3 dairy-type, first-calf heifers of mixed breeding. Heifers 368 and 369 were in about the sixth month of gestation, while the fetus in heifer 371 was nearly full-term. Inoculations were made into the jugular vein, but the doses of organisms varied. Heifer 368 received 38,000,000 organisms of strain 5-59 per kilogram of body weight, from a total dose of 17 ml. of broth culture. Heifer 369 received 36,000,000 organisms of strain 4-59/kg. from 16 ml. of broth culture, and heifer 371 received 30,000,000 organisms of strain 18-58/kg. from 14 ml. of broth culture. Following inoculation, the cattle were observed clinically and tested for antibody response, changes in the leukocyte concentrations, and the presence of the organism.

The agglutination test and antigen-fixation test were used to detect the time and extent of antibody response.[9,11] Changes in leukocyte concentrations were determined by total and differential white blood cell counts.

Blood cultures were made by aseptically withdrawing 4 ml. of blood from the jugular vein and dividing it as follows: Two bovine blood agar plates were inoculated with 1 ml. each and the remaining 2 ml. of blood was placed in a tube of tryptose broth. The latter was held in the refrigerator for 6 weeks, at which time it was recultured. Following abortion, uterine exudates were collected daily from the anterior part of the vagina. These were obtained by inserting a sterilized pipette into the vagina and aspirating with a sterile rubber bulb.[3] Organs and exudates were cultured by means that have already been described.[9] After 6 weeks of storage at 4 C., the specimens were recultured.

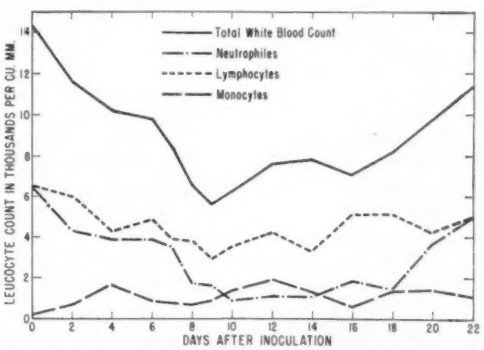

Fig. 1 — Leukocytic changes in pregnant heifers inoculated intravenously with *Listeria monocytogenes*. The plots represent mean counts for heifers 368 and 369.

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Results

Clinical Events.—The heifers aborted and their clinical behavior up to the time of abortion was similar. High body temperatures during the first 48 hours following inoculation ranged from 102.2 to 104.0 F. There seemed to be little impairment of appetite during that time, but inappetence was noted on the fifth day. The cattle then had preparturient development in the mammary glands and edema of the vulva. Heifers 368 and 371 aborted on the sixth day, and heifer 369 aborted on the eighth day following inoculation. Events varied somewhat following the abortions, necessitating consideration of individual animals.

The fetus of heifer 368 was in approximately the sixth month of gestation and was dead when expelled. Fetal cotyledons were brown; there was abnormal opacity to the chorioallantois.

The heifer incurred systemic effects from the Listeria infection. The temperature was 102.0 F. on the day of abortion, rising to 105.2 F. 48 hours later, at which time the heifer was dyspneic and recumbent. The fetal membranes were firmly attached and no effort was made to remove them or to medicate the uterus. Her temperature remained above 102 F. for 7 days following abortion and, since she ate meagerly during that period, there was considerable loss of condition. The fetal membranes remained hanging for 10 days and then began to shred, with fragments passed as late as postabortion day 28.

A genital examination made 11 days after the abortion revealed a normal rate of uterine involution. Within a day following abortion, the uterine discharge was in transition from a watery nonodorous fluid to opaque pus with a fetid odor. This transition was associated with the change from a specific Listeria metritis to an organ that was massively invaded by secondary microbial populations entering from the exterior. Copious quantities of pus persisted until postabortion day 19 when normal mucus was observed mixed with the pus. Twenty-eight days after the abortion, a small amount of pus was still evident. The cow began to regain weight and appeared in good health 4 months after the abortion.

Uterine exudate was collected daily following abortion and examined bacteriologically (table 1). The Listeria organism was isolated every day for 13 days. Eight more samples examined beyond that day were uniformly negative. The last sample was collected 28 days after abortion. Blood cultures were made daily for 10 days beginning 24 hours after inoculation. These were uniformly negative. Colostrum-like secretion from the mammary gland was cultured on 5 separate days. It yielded Listeria organisms on the first and second days after abortion, but not on the third, sixth, and seventh days.

The fetus of heifer 369 was also estimated to be in its sixth month of gestation and was born dead. It was collected while passing through the birth canal, after which the heifer expelled great quantities of fetal fluids. They were nonodorus and had the same appearance as the fluids from heifer 368. A sample of allantoic fluid was collected and enumerated quantitatively by the plate-count method to determine the degree of contamination occurring at the time of Listeria abortion. The fluid contained 9.8×10^8 Listeria cells per milliliter. The fetal membranes had the same appearance as those seen from heifer 368.

Abortion occurred 8 days ater inoculation, at which time the temperature was 103.4 F. The heifer preferred to lie down before and after the abortion. Three days after expelling the fetus, the heifer would stand and eat, although considerable loss of condition was apparent. Her temperature did not go above 102.5 F. for 20 days following abortion, but during that time there were 9 readings of 102.0 F. or higher. The fetal membranes remained attached and were visible at the vulva for 8 days.

An examination on postabortion day 9 revealed a normal rate of uterine involution. Blood cultures were made daily for 10 days following the Listeria inoculation. The only positive culture was obtained 24 hours after inoculation (table 1). The milklike secretion from the mammary gland was positive for Listeria on postabortion day 4, but not on days 1 and 5. The genital exudates were similar to those already described and were cultured daily.

Listeria monocytogenes was recovered each day for 9 days. After that, 10 more culture attempts were made which were uniformly negative. Pus was still evident 32 days after the abortion, but not at 36 days when the heifer was killed and necropsied. The heifer was examined because

she failed to return to normal condition. Complications became apparent 20 days after abortion when intermittent fever occurred, along with leukocytosis and fluctuating evidences of weakness and inappetence. There were no signs of encephalitis, but the circumstances warranted a search for persistent foci of Listeria infection. Necropsy disclosed an unanticipated pyelonephritis which was severe enough to explain the later clinical signs. Extensive bacteriologic examination of the kidney, urine, uterus, and iliac lymph node revealed *Corynebacterium pyogenes*. In addition, *L. monocytogenes* was found in the kidney and urine (table 2).

The genital tract of heifer 371 was palpated 5 days after inoculation and the fetus was found to be alive. At that time, activity in the mammary gland and other signs indicated an impending abortion. Expulsion of the fetus began during the night, but the large, fully formed Holstein-Friesian fetus became impinged in the birth canal and was not removed from the heifer until noon of postinoculation day 6. A few hours later, the heifer stood up briefly and then again went down. She was treated in a recumbent position for 19 days following the abortion. The temperature receded to 99.2 F. on the day of abortion which was probably a reflection of the near fatal termination of the heifer at that time. The temperature was 103 F. on postabortion days 3 and 4. It fluctuated, thereafter, but never exceeded that reading. Antibiotic therapy, begun 24 hours after abortion, included penicillin and streptomycin intramuscularly and oxytetracycline in the uterus. This was continued for 9 days, along with calcium gluconate, glucose, and stilbesterol as supportive therapy. The fetal membranes were retained but were removed manually 4 days after abortion. Uterine exudate yielded Listeria organisms when cultured at 24 hours, but not when again examined 8 days following the abortion (table 1). Seven blood cultures were made from which bacteremia was seen on postinoculation day 1 and on postabortion day 1.

The heifer continued to be recumbent, even though she had sensory and motor

TABLE 1—Isolations of *Listeria monocytogenes* from Body Fluids of Experimental Heifers

Days after inoculation	Heifer 368 Strain 5-59						Heifer 369 Strain 18-58						Heifer 371 Strain 4-59			
	Blood		Uterine exudate		Mammary secretion		Blood		Uterine exudate		Mammary secretion		Blood		Uterine exudate	
	Or.*	Re.**	Or.	Re.	Or.	Re.	Or.	Re.	Or.	Re.	Or.	Re.	Or.	Re.	Or.	Re.
1	−	−	.....	.....	.....	.....	−	+	.....	.....	.....	.....	−	+	.....	.....
2	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	NC	−	.....	.....
3	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	NC	−	.....	.....
4	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....
5	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....	.....	.....
6	−‡	−	+	−	.....	.....	−	−	.....	.....	.....	.....	−‡	−	.....	.....
7	−	−	+	+	NC	+	−	−	.....	.....	.....	.....	−	+	+	+
8	−	−	+	+	NC	+	−‡	−	.....	.....	.....	.....	.....	.....	.....	.....
9	−	−	+	+	−	NC	−	−	+	+	−	NC	.....	.....	.....	.....
10	−	−	+	+	.....	.....	−	−	+	+	.....	.....	.....	.....	.....	.....
11	.....	.....	+	+	.....	.....	.....	.....	+	+	.....	.....	.....	.....	.....	.....
12	NC	−	+	+	−	−	NC	−	+	+	−	+	.....	.....	.....	.....
13	.....	.....	+	+	−	NC	.....	.....	+	+	−	NC	.....	.....	.....	.....
14	NC	−	+	+	.....	.....	NC	−	+	+	.....	.....	.....	.....	−	−
15	.....	.....	+	+	.....	.....	.....	.....	+	+	.....	.....	.....	.....	.....	.....
16	NC	−	+	+	.....	.....	NC	−	+	+	.....	.....	.....	.....	.....	.....
17	.....	.....	+	+	.....	.....	.....	.....	+	−	.....	.....	.....	.....	.....	.....
18	.....	.....	+	+	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
20	.....	.....	.....	.....	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
22	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
23	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
24	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
25	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	−	−	.....	.....
27	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
29	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
30	.....	.....	NC	−	.....	.....	.....	.....	NC	−	.....	.....	.....	.....	.....	.....
31	.....	.....	−	−	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....
34	.....	.....	−	−	.....	.....	.....	.....	−	NC	.....	.....	.....	.....	.....	.....
36	.....	.....	.....	.....	.....	.....	−	−	.....	.....	.....	.....	.....	.....	.....	.....

*=Original culture; **=reculture; ‡=day of abortion; +=L. monocytogenes isolated; −=negative result; NC and ..... =not cultured.

TABLE 2—Isolations of *Listeria monocytogenes* from Experimental Bovine Fetuses and Their Dams

Tissues cultured	Fetuses						Dams			
	Fetus 368 Strain 5-59		Fetus 369 Strain 18-58		Fetus 371 Strain 4-59		Heifer 369 Strain 18-58		Heifer 371 Strain 4-59	
	Or.*	Re.**	Or.	Re.	Or.	Re.	Or.	Re.	Or.	Re.
Medulla	....	....	+	+	+	+	−	−	+	+
Pons	+	+	....	....	+	+	−	−	+	+
Cerebrum	+	+	+	+	....	....	....	....	....	....
Liver	+	+	+	+	+	+	−	−	−	−
Lung	+	+	+	+	+	+	−	−	−	−
Spleen	+	+	+	+	+	+	−	−	−	−
Kidney	+	+	+	+	+	+	+	+	−	−
Stomach contents	+	+	+	+	+	+	....	....	....	....
Peritoneal fluid	+	+	+	+	....	....	....	....	....	....
Pleural Fluid	....	....	....	....	+	+	....	....	....	....
Urine	....	....	+	+	....	....	−	+	−	−
Heart blood	....	....	+	+	+	+	....	....	....	....
Amniotic fluid	....	....	+	+	....	....	....	....	....	....
Placentome	....	....	+	+	+	+	....	....	....	....
Iliac node	....	....	....	....	....	....	−	−	−	−
Uterus	....	....	....	....	....	....	−	−	−	−
Mammary gland	....	....	....	....	....	....	−	−	−	−
Bile	....	....	....	....	....	....	−	−	−	+
Duodenal contents	....	....	....	....	....	....	−	−	−	+

* = Original culture; ** = reculture; + = L. monocytogenes isolated; − = negative result.

function in all limbs. There were no obvious evidences of cranial nerve dysfunction, although she would eat only when feed was placed in her mouth.

On postabortion day 19, the heifer was killed for necropsy. At that time pus was still being formed in the genital tract. The only gross lesions were a few widely scattered foci in the liver that appeared to be abscesses 2 to 3 mm. in diameter surrounded by a hyperemic border. Bacteriologic examination disclosed Listeria organisms in the bile and duodenal contents which were probably associated with the liver lesions (table 2). The block of liver tissue cultured did not yield Listeria organisms. Listeria cultures were obtained from the medulla and pons of the brain. These isolation were complemented by histopathologic lesions of a mild encephalitis.

Leukocytic Changes.—The combined leukocyte counts for heifers 368 and 369 are shown because they nearly parallel each other (fig. 1). The leukocyte counts were somewhat above expected normal values initially. The count declined to 5,650 cells per cubic millimeter 9 days following intravenous inoculation and remained below the normal mean value of 7,800 cells/cmm. for a total of 10 days.[3] This was the period of metritis associated with retained fetal membranes and pus in the uterine exudates from which a variety of bacteria, in addition to Listeria, could be cultured. The depression in the total number of white blood cells was principally concerned with the neutrophils (fig. 1).

There was a shift to the left with nonfilamented neutrophils, ranging from 1,650 to 2,520/cmm. of blood, appearing 1 to 2 days after the inoculation. Counts on nonfilamented neutrophils remained well above normal values throughout the disease process. A mild monocytosis developed 2 to 3 days after inoculation and persisted throughout the periods of observation. A decrease in eosinophils and some fluctuation in lymphocyte numbers, with slight depression following abortion, also occurred.

Heifer 371 had similar blood changes except for some variation in the neutrophil response. A marked neutrophilia developed 24 hours after inoculation (14,200 neutrophils/cmm. of blood of which 28% were nonfilamented). The neutrophil count quickly fell to a value of 5,300/cmm. of blood on the day of abortion. The count remained near this value for the following 10 days, after which the neutrophils began to increase and reached 7,450/cmm. of blood at the time the heifer was killed, 25 days after inoculation. This heifer was different from the other 2 in that she experienced considerable trauma in delivery and also developed Listeria encephalitis.

Antibody Response.—The agglutinating antibody titers at the beginning of the experiment were 1:200 for 2 heifers and

1:400 for the third heifer. These titers represented the commonly encountered antibodies found in the serums of man and many species of animals. They are usually assumed to be "normal" antibodies and their titers serve as a base line from which to measure true immune antibody response.[11] Serums were collected each 24 hours in order to detect the first appearance of antibody response. Titers began to increase on postinoculation day 4 for 1 heifer and on day 5 for the other 2. Antibodies accumulated rapidly, reaching their peak titers of 1:3,200 and 1:6,400 within 3 to 4 days from their appearance. These titers represented a 4- to 5-fold increase in antibody titer as a result of the inoculation of Listeria cells. Abortions occurred just before or at the time of antibody peak. Titers receded surprisingly soon, so that preinoculation titers were reached in heifers 368 and 369, after 34 and 30 days had elapsed, respectively, following inoculation. The titer of heifer 371 had fallen to 1:1,600 at the time of necropsy, 25 days after inoculation.

The antigen-fixation test became positive 4 to 6 days after inoculation. The test became positive on the day that heifer 371 began aborting its live fetus, but was positive for 48 hours before the other heifers expelled their fetuses, which had died *in utero*. These tests remained positive to the termination of the experiment, even after agglutinin titers had returned to their preinoculation levels. When tested 4 months after inoculation, the test was still positive in heifer 368.

Fetal Studies.—The fetuses were subjected to pathologic and bacteriologic examination on the day they were aborted. The fetus of heifer 371 was fully formed and had extensive edema and hemorrhage around the head and neck. The heifer had been in labor several hours with only the head and 1 foot of the fetus protruding from the vulva. Changes in the fetal tissues indicated that the fetus was alive at the time it entered the birth canal even though the lungs had never been aerated. The only other gross change observed was a beginning autolysis of the kidneys. Abomasal contents were pale yellow in color and contained much flocculent material.

The other 2 fetuses were in about the sixth month of gestation and had been dead *in utero* for an unknown length of time so that autolysis was considerably advanced. There were no gross changes that could be recognized as antemortem lesions. Tissues were stained a pink color and the abomasal contents were likewise reddish, watery fluids containing flocculent particles. The fetuses did not have an offensive odor.

Histopathologically, foci of necrosis could be seen in the livers of all of the fetuses and bacteria could be seen in association with these necrotic areas. Similar foci were seen in the spleen of the fetus from heifer 371. The brains did not have lesions referable to Listeria. Placentomes had neutrophilic infiltrations with some necrosis in the villi and occasional colonies of bacteria in the crypts at the site of union between maternal and fetal tissues.

Bacteria comparable in morphology with Listeria were observed in tissues such as lung, liver, placenta, kidney, spleen, and within the blood vessels of the brain. The observation of bacteria growing freely in the vessels and tissues, without evidence of tissue reaction, was particularly apparent in the 2 younger fetuses which had been dead *in utero* prior to abortion. The Listeria organism was widely distributed in the fetal tissues (table 2).

Discussion

This investigation indicated that the Listeria strains obtained from bovine genital tracts in 3 naturally infected herds were capable of inducing abortion.[9] Intravenous inoculation of Listeria organisms produced a disease process that was similar in many respects to the field cases. Although the initial multiplication of the organism probably started in the uterine caruncle, the lesions there were not massive nor were they consistent throughout a given tissue section. Failure to encounter an active area would be possible when sectioning an individual placentome, yet the lesions encountered in a placentome from heifer 368 were similar in all respects to those seen in a field case in herd B.[9] Distribution of the agent to the genital tract is, very likely, hematogenous just as it appears to be in invasion of the central nervous system.[2]

Active invasion of the bacteria into the tissues of the fetus occurred. This was followed by expulsion of the fetus from the

uterus either alive or dead, which resembled the field cases. Obvious gross lesions in the fetuses were not seen in either the experimental or field situations. This point is important because the literature frequently mentions gross lesions from listeriosis in young ruminants which leads the diagnostician to expect such lesions from Listeria abortion. When tissues were examined histologically, the expected changes from a Listeria process were seen in the livers and elsewhere.

The sequelae of abortion in the experimental heifers were similar to those observed in field cases. There was apparently enough metritis to cause retention of fetal membranes in all cases. Fever and loss of condition were likewise encountered. Occasional deaths of the dam were reported in field cases. These probably occurred as a result of either metritis or encephalitis. The severity of the uterine infectious process would vary with the kind of microbial population that entered following abortion. This invasion could supersede the existing Listeria metritis in importance to the health of the dam. Certainly a percentage of the cattle would also experience invasion of the central nervous system as was seen in the case of heifer 371. As shown by that heifer, the encephalitic process need not display characteristic clinical signs.

There were at least 2 contributing factors to the changes that occurred in the white blood cells of the experimental heifers; *i.e.*, the Listeria process, and retention of fetal membranes. An increase of neutrophils could be expected following the intravenous inoculation of Listeria organisms, and this probably induced the preabortion neutrophilia which was encountered 48 hours after inoculation of bacteria into heifer 371. The other 2 heifers had increased numbers of nonfilamented neutrophils at that time, even though the segmented forms were decreasing. Neutropenia observed following the abortions was probably concerned with retention of fetal membranes. It has been supposed that neutropenia during that period is due to toxemia, with resultant depression in the numbers of cells formed and released by the bone marrow.[8] The uterine exudates contained enormous numbers of neutrophils which suggested that rapid removal from the circulation may also be a contributing factor. All of the heifers had a recovery in neutrophil numbers about 20 days after inoculation.

A level of monocytosis comparable to that seen in these cattle has been described following retention of fetal membranes.[14] In this study, increased numbers of monocytes appeared prior to the abortions and may be considered a result of the Listeria process at that time, which became complicated with response to the trapped necrotic tissue in the maternal caruncles following abortion. The monocytosis was not of sufficient magnitude to serve as a diagnostic feature for abortion induced by *L. monocytogenes* in cattle.

Bacteriologic examinations in the experimental study were made under ideal conditions wherein the fetuses were collected as they were aborted. All tissues and fluids were harvested with care, to avoid contamination, and cultured immediately. This procedure gave a far more complete picture of the distribution of the agent than was possible in the field cases. The extent to which Listeria is shed following abortion was observed in the experimental study. At the time of abortion the fetal fluids from heifer 369 contained 9.8×10^8 organisms per milliliter. The agent was obtained from uterine exudates of heifers 368 and 369 for 9 to 13 days following abortion. Apparently the uterus then eliminated the infection since further examinations did not again yield Listeria organisms. These events probably play an important role in the epizootiology of Listeria abortion. If one pregnant cow becomes infected, even through indirect contact with Listeria-infected wild animals,[12] it can serve as the infection source to many other cattle which lick the fetus or ingest uterine fluids.

The necessity for reculturing in bacteriologic examinations for Listeria was demonstrated again. Among 63 positive tissues or fluids subjected to dual culturing, 7 (11%) were obtained only on reculture. This is in contrast to the field studies in which 10 isolations were made and 6 (60%) were obtained on reculture.[9] The number of Listeria organisms in the specimen and the extent of contaminating microflora undoubtedly have much to do with the relative importance of the reculture procedure. Many of the field samples were heavily contaminated and the Listeria populations they contained were, no doubt, drastically reduced during the

relatively protracted period between sampling and culturing.

Even though the pregnant bovine host had a prompt immunologic response to the intravenous inoculation of Listeria cells, as evidenced by the appearance of antibodies after 4 to 5 days, the response was unable to thwart the process that had already begun in the uterus. Bacteremia was detected in 2 of the 3 heifers 24 hours after inoculation. The immunologic response may have been responsible for preventing this from occurring extensively since it might be assumed that the uterine lesions could release numerous organisms into the circulation. This was evidenced when a positive blood culture was obtained from heifer 371 at a time of extreme debility the day following abortion.

The rapid decline in antibody titer was surprising not only since the heifers had received antigenic stimulation from the intravenous inoculation, but also since the uterine infection had produced a great amount of additional antigen. The rapid loss of antibody is likewise in contrast to the experience with sheep in which marked increases in titer have persisted 2 years after being inoculated with live Listeria organisms.[10] Although the agglutinin titers had receded to preinoculation levels within approximately 1 month in 2 of the heifers, the antigen-fixation test revealed that a more persistent change in antibody population had, nevertheless, been produced by the Listeria process.

From sporadically performed cultural examinations of mammary gland secretions, the Listeria organism was encountered on the first, second, and fourth days following abortion. This corroborates the findings of others that *L. monocytogenes* can be found in milk from infected cows.[4,13] The importance of this to the epidemiology of listeriosis in man requires further elucidation.

Summary

1) Three strains of *Listeria monocytogenes*, isolated from natural cases of bovine abortion, induced abortion in heifers 6 to 8 days after intravenous inoculation.

2) Retained fetal membranes, fever, neutropenia, and weight loss characterized the clinical picture following abortion.

3) The Listeria organism was isolated from the uterine exudates of 2 heifers for 9 and 13 days after abortion.

4) The organism invaded the fetuses without producing any characteristic gross changes, but lesions could be detected microscopically.

5) *Listeria monocytogenes* was isolated from mammary gland secretions following abortion.

6) Two heifers, killed 25 and 36 days after inoculation, still harbored *L. monocytogenes* in their tissues.

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Eradication Versus Control in

Communicable Disease Prevention

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THE ERADICATION concept of disease prevention requires, for its best application, continuing expansion at the periphery, and across international frontiers. The Pan American Sanitary Bureau has long recognized the interdependence of human and veterinary preventive medicine; the Bureau organized the first official international veterinary public health activities in 1949, planned and created the Pan American Aftosa Center in Rio de Janeiro, Brazil, in 1951, and the Pan American Zoonoses Center in Azul, Argentina, in 1956.

In considering eradication versus control of communicable diseases, the principles involved are the same in dealing with human, animal, and plant diseases, and with the zoonoses which attack both animals and man.

By reversing the dictionary procedure and giving examples of usage before defining terms, the differentiation of eradication and control may be simplified. The first example is from the Annual Report of the Rockefeller Foundation for 1915:[9] "The . . . policy of the International Health Commission (of the Rockefeller Foundation) . . . has been that of demonstrating, in a limited area of each country, the feasibility of bringing disease . . . under control . . . by showing that it is possible to clean up a limited area, an object lesson is given, the benefit of which is capable of indefinite extension." The concept of cleaning up a limited area and extending it indefinitely is the concept of eradication.

Quoting again from the Foundation's 1915 Report:[10] "Preliminary arrangements have been made for a survey to determine the feasibility of undertaking, at this time, the eradication of yellow fever, and for experiments to test the practicability of controlling malaria." Here is a clear-cut differential usage of the terms, the eradication of yellow fever, meaning the world wide eradication of the virus of yellow fever, the local eradication of which had been demonstrated in Havana, Panama, Rio de Janeiro, New Orleans, and elsewhere; and the control of malaria, which was destined to be a frustrating problem in most rural areas of the world until the introduction of DDT, four decades later.

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Another example of usage of the terms "eradication" and "control" is from a recent article by T. Lloyd Jones,[4] "Using methods that make medical health officers envious, veterinarians in several countries have completely stamped out such diseases as rinderpest, contagious bovine pleuropneumonia, glanders, dourine, hog cholera, foot-and-mouth disease, and others, and have brought under control such diseases as brucellosis and bovine tuberculosis, all by the short-cut of slaughtering affected animals." Here is a clear-cut distinction, in referring to past accomplishment, between eradicating and controlling disease, even though there are well-advanced eradication programs for both brucellosis and bovine tuberculosis. These eradication programs are not control programs, and they have done much more than bring brucellosis and bovine tuberculosis under control; they have resulted in establishing areas of local eradication as essential stepping stones to national eradication.

Just as the police officer considers crime to be under control when it is at such low level that it is of importance only to the victims, so the public health officer considers a given disease to be under control once its incidence has been so reduced that it is no longer a serious public health problem except to the few voiceless victims who still suffer. May I assume that in veterinary practice, control means the reduction of a disease until it is no longer a source of serious financial loss to the com-

Fig. 1—Physicians, Department of Health, Veterinarians, and Department of Agriculture work together against the zoonoses.

munity as a whole, although it may be to those whose animals are affected?

Eradication, on the other hand, refers to the complete disappearance of all sources of infection of a given disease agent, so that no recurrence of that disease is possible, even in the absence of all preventive measures. Local eradication indicates the elimination of all sources of infection from a given area, so the disease does not recur unless reintroduced from outside the area.

Eradication Programs

Eradication programs must cover fairly large areas from the beginning to minimize the possibility of reintroductions of the infection into clean areas, and they must be able to expand at the periphery. Local eradication, to be successful, must provide for protection against reinfection. Such protection can be best obtained by increasing the eradication area. Eventually the limits of the eradication area will coincide with the national boundaries. At this point, the nation acquires a vested interest in eradication in neighboring countries. Eradication programs evolve naturally as local, state, national, international, regional, and world programs.

Fortunately for the ultimate development of health programs throughout the world, there is no convenient stopping point; eradication is not something one area can do for itself and maintain indefinitely with ease. It is obvious that clean areas can best be protected by sharing the burden of cleaning up less fortunate areas through pooling financial and technical resources. Only in recent years, with the development of the Pan American Health Organization, the World Health Organization, the Food and Agriculture Organization of the United Nations, the International Cooperation Administration of the U.S.A., and UNICEF, has there existed an adequate mechanism for coordinating and assisting regional and world eradication programs.

The first international and world eradication attempt antedates the effective development of these organizations by 30 years. The survey to determine the feasibility of undertaking eradication of yellow fever, mentioned in the quotation from the "Rockefeller Foundation 1915 Annual Report," led to an attempt to eradicate yellow fever from the world. The Rockefeller Foundation, a private philanthropic organization, coordinated the antimosquito programs of affected countries, and gave the technical and financial assistance needed.

Wickliffe Rose, who, after consultation with William C. Gorgas, committed the Rockefeller Foundation to yellow fever eradication, was without training in medicine and public health administration. Rose had been a professor of philosophy and a college dean before entering the field of public health as executive secretary of the Rockefeller Sanitary Commission for the Eradication of hookworm disease in 1910 and continuing as director of the International Health Commission when the Foundation was chartered in 1913. Fosdick has said of Rose,[9] "Always his thinking and his strategy were in planetary terms. Whether it was mathematics or chemistry or astronomy, he worked on a global scale. His effort was constantly to break over the boundaries of parochialism and lay his plans in accordance with world patterns."

The yellow fever-eradication program is the most magnificent failure in public health history. When the Foundation withdrew from the program in 1949, after 34 years of effort, during which the lives of 6 staff members were sacrificed to accidental infections and some $14 million were spent on the control and study of the disease,[10] yellow fever virus persisted widespread in the forest animals of Africa and South America. But the attempt to eradicate yellow fever led inevitably, as many decades of control might not have done, to studies which clarified the epidemiology and epizootiology of yellow fever and to the perfection of a live attenuated virus vaccine. (The techniques for handling viruses, developed during the studies of yellow fever virus, have been of inestimable value in the study of other virus diseases.)

With the discovery of jungle fever in 1932,[12] it became obvious that the eradication of yellow fever virus had, from the beginning of Rose's dream in 1915, been impossible. But by 1934, the last endemic focus of human yellow fever in the Americas had been cleared of infection. Since 1935, yellow fever in the western hemisphere has behaved essentially as a zoonosis;[17] few urban cases of yellow fever have occurred in the past 25 years, and those have come from transient invasions of cities and towns by the jungle virus brought in by persons infected in the forests. In the future, even this threat to urban communities should disappear; already it has largely disappeared with the progress of the campaign for the eradication of the *Aedes aegypti* mosquito, the only urban vector of yellow fever in the western hemisphere.

This continental campaign grew out of the observation, in 1933, that *Aedes aegypti* had been eradicated from a number of Brazilian cities. This program developed by stages; first the coastal cities of North Brazil, then the suburbs, smaller cities, and even rural areas of Brazil were cleared of *Aedes aegypti*. But the demand for expansion of the eradication program at the periphery for the protection of clean areas has been insatiable. Since 1947, the nations of the Americas, at the insistence of the Brazilian government, have been committed

to the eradication of the *Aedes aegypti* mosquito from the western hemisphere as the only means of guaranteeing the cities and towns of the Americas against reinvasions by the jungle virus.[6] Reports presented to the Fifteenth Pan American Sanitary Conference (Puerto Rico, 1958) show that *Aedes aegypti* is no longer to be found in Bolivia, Brazil, British Honduras, the Canal Zone, Ecuador, French Guiana, Nicaragua, Panama, Paraguay, Peru, and Uruguay. Later reports[7] give clearance to Honduras and Guatemala.

It is interesting that the program for the eradication of yellow fever was undertaken in 1915 on the basis of a philosophical and intellectual approach to the world eradication of the yellow fever virus, supported by the observation that yellow fever disappeared following the reduction in incidence, not the eradication, of the *Aedes aegypti* mosquito.

The first eradication of this species from the capital cities of northern Brazil in 1933[15] came as an unexpected dividend from careful administration of antimosquito measures and not from a conscious effort at species eradication. Once these cities were free of *Aedes aegypti*, it proved to be more economical to clean out the places from which they might be reinfested than to maintain permanent antimosquito measures in the cities themselves.

The development of the administrative techniques which led to the eradication of *Aëdes aegypti* were later adapted to the eradication of *Anopheles gambiae* from Brazil in the memorable campaign of 1939-1942.[14] This eradication of *Anopheles gambiae*, the most dreaded of Africa's vectors of malaria, from its 300 mile-on-a-side triangular bridgehead on the American Continent was of vital importance to the invaded area and to the enormous areas in South Central and North America and in the West Indies threatened by progressive invasion of the species.

The technique used and the experience gained with *Anopheles gambiae* in Brazil were used in the eradication of this same mosquito from Egypt in 1944-1945, three years after a disastrous invasion from the Sudan.[11]

An important result of the successful eradication campaign with *Aëdes aegypti* and *Anopheles gambiae* was the beginning rehabilitation of the concept of eradication among public health workers. The commitment of the Americas to the eradication of *Aëdes aegypti* in 1947 was followed by similar undertaking for yaws, smallpox, and malaria in 1950.[6]

The eradication of smallpox had been feasible for many decades, but the campaigns against yaws and malaria are based on recent developments — penicillin and DDT.

The introduction of DDT and other residual insecticides made the control of rural malaria economically feasible. Following the observation of local eradication of malaria, national and regional eradication programs were undertaken. In 1955, these culminated in the acceptance by the Eighth World Health Assembly meeting in Mexico City of a program for the world eradication of malaria.[16] Today the health authorities of almost all of the malarious countries outside of Africa are engaged in a precedent making world-wide campaign of malaria eradication, designed to end forever this curse. The national programs are being coordinated and assisted by the Pan American and World Health Organizations, by UNICEF, and by the International Cooperation Administration of the United States.

A full quarter of a century before Rose, the philosopher, undertook the eradication of yellow fever, a young man destined to become one of America's great public health leaders, Charles V. Chapin, presented a philosophy of disease eradication as a natural corollary of the acceptance of the germ theory of communicable disease. In 1888, a few years after R. Koch's discovery of the tubercle bacillus, Charles V. Chapin said,[1] "In regard to the prevention of consumption, it must be admitted that the germ theory has done little except to emphasize the importance of hygienic measures. But it should have great influence. . . . The germ theory—now no longer a theory in the case of tuberculous consumption—tells us what we have to do with a contagious disease. Now there is no theoretical reason why a purely contagious disease like tuberculosis cannot be exterminated. If we can prevent the spread of contagion at all, we can prevent it entirely." This last sentence is a fitting slogan for all who are responsible for the prevention of communicable diseases. Once this principle is accepted, the problem becomes one of devising economically feasible administrative methods adapted to each situation. This "economically feasible" restriction is most important; methods chosen must be not only feasible, but economically so.

Eradication programs are based on a variety of measures: for *Aëdes aegypti*, on an attack on the aquatic stages of development; for smallpox, on herd vaccination; for yaws, on mass treatment of cases

and contacts; and for malaria, on the destruction of infected mosquitoes.

Not every disease, nor every disease vector, is presently eradicable; the campaign for the eradication of the urban vector of yellow fever is well advanced, but no one has yet proposed any eradication of the forest mosquito vectors of jungle yellow fever.

Chapin recognized the need of concomitant solution of the problem of bovine tuberculosis. To quote:

> But with the increasing prevalence of tuberculosis among domestic animals something more is imperatively demanded. Active measures should be taken to free the country from animal tuberculosis. The proper authority for dealing with this, as with all other contagious diseases of animals, is the Bureau of Animal Industry of the Department of Agriculture. It is a wasteful method for states to act independently. The powers and expenditure of this Bureau should be greatly increased and it should take active measures against this disease.
>
> The exact measures suggested are:
>
> 1) The reporting of all cases of tuberculosis in domestic animals to the proper authority by both owners and veterinarians or other persons having a knowledge of the same.
>
> 2) The slaughter of all infected animals and the isolation and slaughter of all exposed to infection. The Government should partially indemnify all owners of slaughtered cattle.
>
> 3) Thorough disinfection of all buildings occupied by diseased cattle.
>
> 4) The confiscation of the flesh and milk and milk products of all tuberculous animals.

Chapin had the vision and the voice of a prophet; he recognized instinctively that the rejection of spontaneous generation and the acceptance of the germ theory of disease implied the concept of contagious disease eradication. Were Chapin alive today he would be emphasizing that it is a wasteful method for nations to act independently, and he would be championing the cause of world-eradication programs.

Only recently did I learn (from Dr. H. C. King) that the Bureau of Animal Industry, which Chapin called upon to eradicate bovine tuberculosis, had been created only 4 years before (1884) specifically for the "suppression and extirpation of contagious disease among domestic animals." To quote Dr. King:

> In 1843 . . . contagious pleuropneumonia gained entrance to the United States. . . . For the next 40 years, this disease spread up and down the Atlantic Coast. Other diseases began to spread and become increasingly alarming. Boycotts by Great Britain against American livestock aggravated growing livestock disease problems, and added economic pressure to the desire by the people that Congress do something about the situation. As a result there was passed by Congress on May 29, 1884, 'An Act for the establishment of a Bureau of Animal Industry to prevent the exportation of diseased cattle and to provide means for the suppression and extirpation of pleuropneumonia and other contagious diseases among domestic animals.'
>
> Five years later, contagious pleuropneumonia had been eradicated (from the U. S.) with the expenditure of $1½ million.
>
> The eradication of contagious pleuropneumonia was a dramatic illustration of what could be accomplished through unified activities in animal disease eradication. The principles involved have been applied successively to a number of zoonoses with gratifying results. Many benefits have accrued to the people of the United States through the suppression of diseases which are transmitted from animals to man. Animal husbandry, as we know it today, with its concentration of animal populations and efficient, rapid, long-distance transportation of livestock, would be practically impossible, had not these diseases been eradicated or effectively controlled. As new tools and methods for disease eradication became available more diseases were included in programs designed to wipe them out. Accomplishments in programs aimed toward the eradication of tuberculosis, brucellosis, cattle tick fever, vesicular exanthema, foot-and-mouth disease, scrapie, and screwworm, are worthy of review and comment.

It is not only the public health authorities who today are thinking in terms of world disease eradication. The Food and Agriculture Organization / OIE Animal Field yearbook for 1957 states,[2]

> It has often been said that diseases know no frontier; regional control of infectious diseases, therefore, is an objective which must receive full consideration and support . . . an example is rinderpest which, although its ravages have been widespread in the past, is now confined to a few areas, and even in them is gradually being eradicated. . . . One of the main objectives of international organizations interested in livestock production is to encourage and participate in the regional control of epizootics leading to their final eradication, not only from the regions but from the world. This is a task fraught with many difficulties; progress, however, is gradually and surely being made.
>
> In the control of diseases of animals, full consideration has to be given to that important group now termed, zoonoses, in which both animals and human beings are involved . . . in such diseases the simplest and probably the most effective measures of control concern the eradication of the infection from the animal populations. There must, therefore, continue to exist close collaboration between veterinary and medical authorities, whose common objective should be to so interweave their activities that zoonotic diseases will eventually be well controlled and even eradicated. In this work veterinary authorities have an important part to play.

However, if they are to play this part, the leaders of the veterinary profession must recognize their responsibility in pro-

moting and advancing the philosophy of communicable disease eradication; the schools of veterinary medicine must emphasize the teaching of the basic elements of epizootiology and of the prevention of communicable diseases; veterinary authorities must accept Chapin's dictum: "If we can prevent the spread of contagion at all we can prevent it entirely." The veterinarian must join the physician in showing more concern over the gap between current incidence of a disease and the zero baseline, than in satisfaction in such reduction of disease incidence as may have occurred. In taking credit for such reduction, the public health officer and the animal health officer must accept blame for what remains. To the eradicationist, the demonstration of his ability to reduce the incidence of a disease constitutes proof of his culpability in not eradicating it. In determining eradication programs for certain diseases, especially the zoonoses, the veterinary health officer must be ready to substitute humanitarian consideration for financial consideration.

Finally, the veterinary health authorities of each country must get their respective governments to accept the responsibility, as members of the community of nations, to prevent their territories from becoming or remaining sources of infection for countries from which a disease has been eradicated, although the disease in question may be relatively unimportant in their countries.

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Clostridial Mastitis Alleviated with Antibiotics

Mastitis in the right front and rear quarters of a pregnant heifer was found to be due to *Clostridium perfringens* and *Escherichia coli*. The udder was warm and swollen. The secretions contained blood and gas. Subcutaneous crepitation could be palpated up to the back of the cow. Recovery followed treatment with 5 million units of penicillin and 8 Gm. of streptomycin for 6 days systemically. Both quarters were infused with oxytetracycline. Both right quarters were lost.—*Tijdschr. v. Diergeneesk., 85, (1960): 79.*

Injectable Tranquilizers

for Weaning and Shipping Calves

D. C. CLANTON, PH.D.
J. K. MATSUSHIMA, PH.D.

IN RECENT years, tranquilizers have received considerable attention regarding their possible use in various phases of cattle management. The use of injectable tranquilizers has been suggested to prevent shrinkage in cattle while in shipment, to reduce stress at weaning time or any other time involving an environmental change, and to quiet cattle while they are being restrained for diagnosis or treatment of disease.

Injectable tranquilizing compounds have produced variable behavior responses in beef cattle.[5] To achieve the same quieting response, nervous, wild calves have required larger doses of tranquilizing compounds than docile calves. Indications are that it would be difficult to determine dosage rates for cattle of different ages, weights, and temperaments.

In a feedlot-adaptation test of yearling cattle, intramuscular injections of 25, 50, and 75 mg. of perphenazine per head did not improve gains or feed consumption the first 14 days in the feedlot.[6]

Pretreatment of feeder cattle destined for shipment, with 75 to 175 mg. of perphenazine, reduced shrinkage and disease incidence and facilitated cattle handling during shipment.[1,4] Yearling feeder cattle given 37.5, 84.0, and 93.0 mg. of perphenazine in the afternoon had a greater overnight shrinkage than nontranquilized cattle, but their shrinkage was less the next day while in transit.[6] Over-all shrinkage was greater in the tranquilized cattle. Shrinkage recovery patterns were about the same for the tranquilized and nontranquilized cattle. In a preliminary report, tetrahydrozoline and isobutrazine injected 30 hours before shipment were effective in reducing shrinkage on fat cattle which were hauled 110 to 120 miles to market.[2] Tissue damage resulted from the injection; there was no difference in dressing percentage.

Tranquilizers have a place in cattle management when it is desirable to quiet them for handling purposes.[3,7]

The value of using an injectable tranquilizer at weaning time was investigated on a typical Sandhills ranch in Cherry County, Nebraska. The objectives of the experiment were to investigate practices and methods of management which would reduce weight loss and disease incidence at weaning time.

Experimental Procedures

In October, 1958, 132 heifer and steer calves were individually weighed and identified at weaning time.

As they crossed the scales, every other calf was given, subcutaneously, 60 mg. of trifluprom azine* in paste form. The calves were observed closely for several days and reweighed 8 days after weaning. During this time, the nontranquilized and tranquilized calves were in a common corral. Because of limited corral space, it was impossible to separate them.

The following fall (1959), the treatment with injectable tranquilizers was repeated. The same procedure was used as in the previous year except that the nontranquilized and tranquilized calves were kept in separate corrals to rule out the effects the tranquilized and nontranquilized calves may have had on each other.

Small differences were observed in the incidence of disease between the tranquilized and the nontranquilized calves. The temperate weather during the week following weaning in 1958 may have contributed to a low incidence of disease. Less

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The cooperation of Forrest Lee, 47 Ranch, Brownlee, Neb., made this experiment possible.

*Trifluprom azine was furnished by E. R. Squibb and Sons, New York, N.Y.

than 3 per cent of the calves in both lots had signs of disease during the experimental period. In 1959 at weaning time, it was cold, windy, and dusty. There was a high incidence of disease characterized by fever, mucous discharge, and droopy ears. These signs were observed in 20 per cent of the nontranquilized and 13 per cent of the tranquilized calves. All were listless and ate little. No difference in weight changes were observed (table 1).

TABLE 1—Effect of Tranquilizer on Weight Changes of Calves from Weaning to 7 or 8 Days Postweaning

Groups	1958		1959	
	No. of calves	Av. gain (lb.) (8 days)	No. of calves	Av. gain (lb.) (7 days)
Tranquilized	65	17.9	61	1
Nontranquilized	67	16.2	61	−1

The high gains following weaning in 1958 were not necessarily body tissue gains, because the initial weight was determined after the calves had been separated from the cows and corraled early in the morning. There was time for a 5- or 6-hour shrinkage. The second weight was taken in the morning after the calves had been fed grain and given access to hay during the night. Therefore, it is assumed that most of the gain was due to fill. Because of inclement weather and the relatively high incidence of disease during the postweaning period in 1959, the calves did not gain weight.

A quieting effect was observed in the tranquilized calves for approximately 36 hours, after which time it was difficult to tell which had been tranquilized. It appears that practices which involve the handling of calves and getting them accustomed to people, corrals, hay, and grain before weaning is desirable. All were docile and quiet in the weaning corrals. With the exception of the inclement weather in 1959, weaning conditions were favorable. These results agree with those of other experiments in which different tranquilizer compounds were used.[5,8]

Another test designed to evaluate the effect of trifluprom azine on shrinkage of cattle during shipment was conducted in October, 1958. A total of 180 steer calves were separated from the cows one day and shipped by truck (45 calves per truck) 250 miles the following day. Prior to loading, each calf in 1 truck was given, subcutaneously, 60 mg. of the drug in paste form; in a second truck, each was given, subcutaneously, 60 mg. in liquid form. Calves in the other 2 trucks were not tranquilized. The calves were group-weighed before and after shipping. The weight shrinkage of those given tranquilizer in paste form and the average of those given no tranquilizer were the same. Calves given the drug in liquid form had the least shrinkage. There was a large, unexplainable difference in the 2 nontranquilized groups (table 2).

TABLE 2—The Effect of Injectable Tranquilizer on Shrinkage of Calves in Shipment

	Drug			
	Paste*	Liquid*	Control A	Control B
No. steers	45	45	45	45
Initial wt.	18,670	18,505	18,345	18,015
Final wt.	17,940	18,010	17,480	17,430
Shrinkage (lb.)	730	495	865	585
Shrinkage (%)	3.91	2.67	4.72	3.25

*Subcutaneous injections of 60 mg. of trifluprom azine just prior to shipment.

Liquid and paste forms of tranquilizer differ in rapidity of absorption in the tissue, the liquid form being more rapidly absorbed.

Conclusions

There is need for further study of dosage levels and methods of administering tranquilizers. The age, weight, and temperament of cattle, as well as anticipated stress or environmental change must be considered. Variation in these factors accompanied by a lack of standardized treating and weighing procedures have probably contributed to controversial results reported by different research workers.

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Bovine Mastocytoma

— A Case Report

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MASTOCYTOMAS occur frequently in dogs but are rare in other species. They are composed of masses of mast cells and are characterized by local malignancy with infrequent metastases. These mast cells are relatively large cells derived from the mesenchyma and are located in the connective tissue throughout the body, especially in the skin and liver of dogs. The nucleus of the mast cell is round or oval and the acidophilic cytoplasm contains large granules that stain metachromatically with aniline dyes. The mast cell resembles the basophilic leukocyte, but there is evidence that they are not identical. The function of the mast cell is not clearly understood, although it has been shown that they contain heparin and are rich in histamine.[11]

Literature

Canine mast-cell tumors were first described in 1908.[9] In 1942, another investigator[1] presented a detailed description of a group of these tumors in dogs and proposed the name "mastocytoma." Since that time, many reports have appeared in the literature describing canine mastocytomas. An examination of 709 skin tumors from dogs showed[3] that 29 were mast-cell tumors. In a report[8] of the examination of 151 round-cell neoplasms from dogs, 78 were found to be mast-cell tumors. In another investigation,[10] 100 canine mastocytomas comprised 20 per cent of the cutaneous neoplasms studied.

Reports of mast-cell tumors in other species of animals are rare. An ulcerating neoplasm from the lip of a cat has been described.[7] This neoplasm was composed of large mononuclear cells with metachromatic granules in the cytoplasm that were consistent with those described in reports of mastocytoma. References[1] have been cited for a mast-cell granuloma from the lip of a horse and mast-cell hyperplasia in the skin of mice. On the basis of 3 cases accessioned in the American Registry of Veterinary Pathology,[12] it was stated that mastocytomas occur rarely in the skin and omentum of bovine animals. Two cases of mast-cell tumors have been reported[3] as occurring in the subcutaneous tissue of cattle. In 1 case, the lesion was limited to a solitary nodule, while in the other, lesions were widely distributed. In a later article,[4] this same investigator described 5 cases of cutaneous mast-cell tumors in cattle. In 1 of these cases, mast cells were found in the prescapular, iliac, and supramammary lymph nodes; and in another case, nodules were found in the spleen, omentum, mesentery, and kidney. Also described were 2 cases of internal bovine mast-cell tumors not involving the skin. The tumor was found in the thoracic cavity in 1 ani-

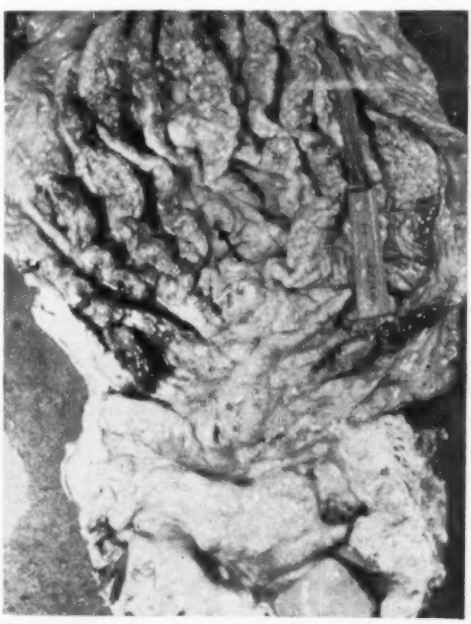

Fig. 1—In a view of neoplastic abomasum of the cow, ulceration (arrow) and thick nodular folds of the mucosa are evident.

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Approved by the Committee on Publications, publication No. 854.

mal, while tumors were found in the lung, uterus, peritoneal cavity, and several lymph nodes in the other animal. A case of bovine mast-cell tumor was reported[2] to the American College Of Veterinary Pathologists. The lesions consisted of multiple cutaneous nodules varying in size up to 2 cm. in diameter with enlargement of some of the superficial lymph nodes. Nodules similar to those found in the skin were present in the spleen, kidneys, liver, and mammary gland.

The purpose of this paper is to describe a mast-cell tumor from the abdominal viscera of a cow.

History

A mature Hereford cow was admitted to the large animal clinic, School of Veterinary medicine, Auburn University, for diagnosis and treatment in September, 1959. The cow had aborted at approximately the sixth month of gestation. Along with 9 others in the herd, it had a high antibody titer for Leptospira. The principal clinical signs were anemia, inappetence, diarrhea, and loss of weight. The packed erythrocyte volume was 13 per cent, in contrast to a normal of approximately 35 to 45 per cent. The clinical signs persisted in spite of treatment, and the cow died after 9 days.

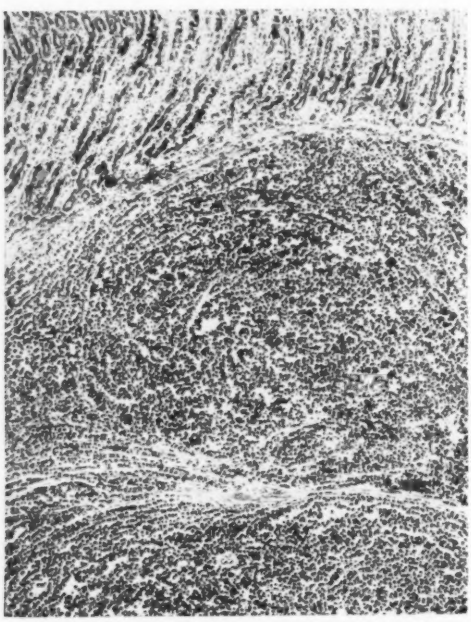

Fig. 2—Abomasal mucosa and submucosa have the lobulated pattern of the mastocytoma. Giemsa's stain; x 43.

Gross Lesions

At necropsy, there was evidence of marked emaciation with serous atrophy of the remaining subcutaneous fat. The integument and superficial lymph nodes appeared normal, and no careful detailed examination of the skin and subcutis was made in an effort to determine if small lesions were present. In view of the final diagnosis this would have been desirable. The only lesion observed in the thoracic cavity was extensive interstitial and parenchymal emphysema of the lungs. This was attributed to agonal struggle.

Examination of the abdominal viscera revealed a greatly enlarged abomasum, the wall of which was extensively invaded with a gray-cream-colored tissue, similar to that commonly seen in cases of malignant lymphoma. The thickening of the wall was so marked that the lumen of the pyloric portion was nearly occluded. The folds of the fundus displayed marked nodular thickening (fig. 1). Many shallow ulcers were present in the mucosa of fundic and pyloric regions. There was generalized hyperemia of the mucosa. After the small amount of feed and sand was washed from the abomasum, it weighed 24 lb. The other compartments of the stomach appeared normal. The rumen contained only a small amount of fluid and partly digested hay.

The abomasal lymph nodes were enlarged and firm, and a few contained areas of necrosis with some calcification. A small, nodular mass at the base of the spleen appeared to be a lymph node invaded by the neoplastic tissue, with considerable necrosis and calcification. No other gross lesions were observed.

In view of the fact that the gross lesions were indistinguishable from those seen frequently in malignant lymphoma, this diagnosis was made, and a single section of tissue was taken from a fold of the abomasum for confirmation. This tissue was fixed in 10 per cent neutral formalin, and paraffin sections were stained routinely with hematoxylin and eosin.

Microscopic Lesions

Evidence of slight edema and proliferation of connective tissue was observed in the mucosa. In some areas, the mucosa had undergone necrosis with a loss of differential staining, but this change was confined to the surface epithelium between the

gastric pits and did not extend to the muscularis mucosae. Small numbers of eosinophils were present, and a few mast cells were found adjacent to the small blood vessels in the mucosa. Lymphocytes were present in areas of the mucosa immediately above the muscularis, and a few mature lymphocytes were found extending into one area of the submucosa.

The tissue section presented an excellent cross section of the submucosa for study. The submucosa was approximately 1 cm. thick and consisted almost entirely of large, round cells with acidophilic cytoplasm and some connective tissue stroma and smooth-muscle cells which separated the closely arranged masses of cells into a lobulated pattern (fig. 2). In two areas, the continuity of the muscularis mucosae had been disrupted, and the cells appeared to be invading the mucosa (fig. 3).

Each of these cells contained a single round or oval nucleus with either 1 or 2 nucleoli. The nucleus was eccentrically located in most cells. Large eosinophilic granules were seen in the cytoplasm, and the cell wall was well defined. Mitotic figures were not seen, but a few of the cells appeared to contain 2 separate nuclei and abundant cytoplasm suggesting recent mitosis. Numerous eosinophils were present throughout the submucosa.

In Giemsa-stained sections, the large cells of the submucosa contained purple granules that completely filled the cytoplasm and presented a typical appearance of mast cells (fig. 4). Some of the cells appeared to have ruptured, and free granules were present in the immediate area. The other cellular elements stained in a normal manner. Tissue sections stained with toluidine blue,[6] Bismark brown,[12] and Unna-Pappenheim's stain[5] also demonstrated the metachromatic characteristics of the cytoplasmic granules.

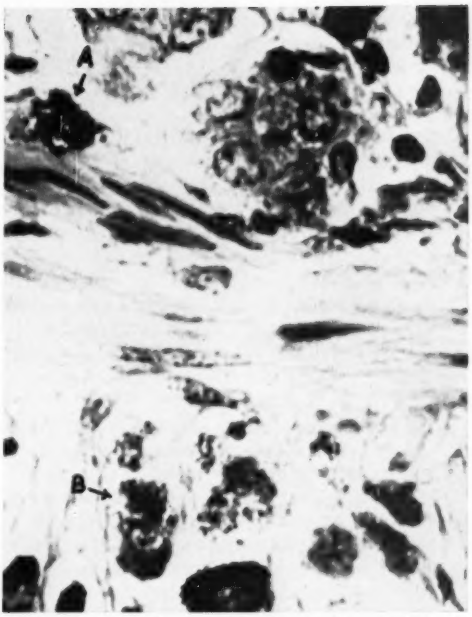

Fig. 3—In a section of the abomasal muscularis mucosae, there were mast cells in the mucosa (A) and the submucosa (B). Giemsa's stain; x 870.

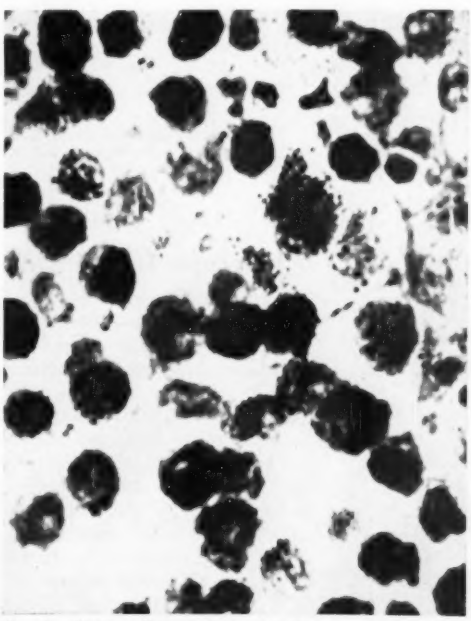

Fig. 4—Mast cells can be seen in this detail of the abomasal submucosa. Giemsa's stain; x 870.

Discussion

Mast-cell tumors occur frequently as skin neoplasms in the dog. In a study of 100 canine mastocytomas, it was found that internal tumors did not occur in absence of cutaneous lesions.[10] The neoplasm described in this report was apparently limited to the abdominal viscera; no cutaneous neoplasms were observed. There was a striking resemblance in color, consistency, and invasive characteristics of this tumor to malignant

lymphoma, a common neoplasm of cattle which frequently involves the wall of the abomasum. Generalized lymphoid proliferation is often found with malignant lymphomas, but it is not too unusual to find the neoplasm restricted to the same tissues as were affected in this cow. It is possible that other tumors of the bovine abomasum which have been diagnosed as malignant lymphoma on the basis of gross lesions may have been tumors of other cell types. This fact emphasizes the need for microscopic examination even though gross lesions appear to be sufficient for a diagnosis.

Summary

1) There are many reports in the literature of canine mastocytoma, but reports of this tumor from other animals are rare.

2) A bovine abomasal neoplasm was diagnosed as a mastocytoma. The gross lesions consisted primarily of marked thickening of the wall of the abomasum and enlargement of the abomasal lymph nodes. Examination of tissue sections of the abomasum showed extensive infiltration of the submucosa with mast cells.

3) Previous reports indicate that mastocytoma in the dog and other animals is usually limited to the skin, but internal metastases occur. In this case, the tumor appeared to be confined to the abdominal viscera.

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Microscopic Test for Antibiotics in Milk

In a microscopic method for detecting antibiotic activity in milk, *Streptococcus thermophilus* culture is exposed to the milk samples for 60 to 90 minutes at 37 C. Milk films are stained with methylene blue and examined for changes in morphology or reduction in clump count as compared to a control in antibiotic free milk. Abnormal enlargement or elongation of cells or a 50 per cent reduction in clump count as compared with the control indicates antibiotic activity. Minimum concentration of various antibiotics detected are penicillin, 0.015 units/ml.; bacitracin, 0.01 units/ml.; oxytetracycline, 0.15 μg./ml.; chlortetracycline, 0.15 μg./ml.; and streptomycin 0.75 μg./ml.

The test procedure is not affected by normal residues of sanitizers or bacteriophage. The stained milk films can be kept as a permanent record. Leukocyte counts can be made on the stained milk films during microscopic examinations.—*J. Milk and Food Tech. (April, 1960): 117.*

Cosmetic Dehorning

D. F. WALKER, D.V.M.

THE FOLLOWING technique of dehorning is not intended to replace ordinary methods used on a herd basis, as it would be too costly and time consuming. This method may be desirable and appreciated by the client in dehorning 4-H calves, steers and heifers to be shown in fat classes, removing broken horns on breeding animals, dehorning in the face of adverse weather conditions, and to avoid sinusitis. Cattle dehorned by this technique are ready for show within a few days following surgery.

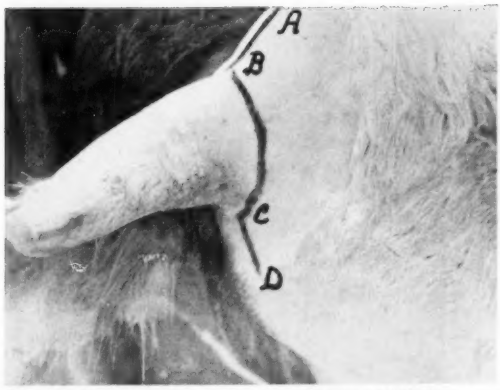

Fig. 1—Skin incision lines are shown (ABCD). Line B-C extends behind the horn as well as in front.

Fig. 2—Skin flaps are reflected and the saw is aligned prior to amputation of the horn.

Fig. 3—A triangular wedge of skin is removed so that the posterior and anterior flaps fit together neatly.

Method

The animal is confined in a stock. One of the tranquilizing agents may or may not be used, depending on circumstances. The poll area is clipped with scissors or clippers. Since we are striving for "first-intention" healing, the area should be thoroughly washed and prepared for aseptic surgery. Anesthesia is acquired by blocking the corneal branch of the lacrimal nerve.

Instruments required are a Bard-Parker knife handle No. 4 with a No. 33 blade, scissors, thumb forceps, 6 towel forceps, 3 to 6 hemostatic forceps, a needle-holding forceps, 2 dermal suture needles, 1 needle suitable for tying ligatures, 1 tube of No. 1 medium chromic catgut, 36 inches of non-capillary No. 3 dermal-type suture, a dehorning saw, and a drape. The drape is placed over the poll and held in place with 2 towel forceps. A skin incision is made starting 1 inch from the midline of the poll and extending along the dorsal border of the head to the base of the horn (fig. 1, line A-B). A 2-inch incision is made, starting at the ventral base of the horn and

The author is associate professor of large animal Surgery and Medicine, School of Veterinary Medicine, Auburn University, Auburn, Ala.; paper No. 857.

Fig. 4—The completed operation with sutures in place, results in a smoothly formed poll.

extending ventrally (fig. 1, line C-D). A skin incision circumscribing the horn is then made, leaving as little skin as possible on the horn (fig. 1, line B-C).

Two flaps, anterior and posterior, are then formed by undermining the skin on the frontal bone and that area between the horn and base of the ear. Large hemorrhaging vessels are clamped with hemostats during this process. A large area is thus exposed at the base of the horn by folding the anterior flap forward and the posterior flap backward. Each flap is immobilized with towel forceps. The horn then may be sawed off with a heavy-backed saw, sawing from anterior to posterior (fig. 2). Use of a surgical wire saw simplifies sterilization; however, the author feels a better job of forming the poll can be achieved with a solid-blade saw.

A wedge or triangular area of skin is removed from the flap at each corner of the incision so that it may be sutured neatly (fig. 3). The exposed area is carefully swabbed to remove bone debris. Hemorrhaging vessels are ligated. The skin is sutured with simple interrupted sutures. Sutures should be left in for 10 days.

Care must be taken when removing the second horn so that the head shape will be the same on both sides (fig. 4).

Antibiotics Given by Many Routes Affects Market Milk

The following recommendations for withholding milk from market following antibiotic treatment of milk cows are proposed in Ohio:

Udder Infusions.—Penicillin, aqueous base—withhold milk from all quarters at least 72 hours; penicillin, oil base—withhold milk from all quarters at least 8 days; penicillin, combined aqueous and oil base—withhold milk 8 days or according to label instructions; oxytetracycline or chlortetracycline, aqueous or oil base—withhold milk from all quarters for at least 6 days; other antibiotics or drugs—withhold milk according to label instructions.

Untreated Quarters.—Antibiotics can appear in untreated quarters of an udder after 1 or more quarters are treated. Therefore, when 1 quarter is treated, the milk from all quarters must be withheld.

Intramuscular or Intravenous Injections.—Milk should be withheld a minimum of 5 days or longer, depending upon circumstances.

Feed Supplements.—Antibiotic-containing feeds are not recommended for dairy cows.—*Anim. Dis. Trends, 7, (April, 1960): 2.*

A Disorder Resembling

Hemophilia B (Christmas Disease)

in Dogs

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J. F. MUSTARD, M.D., PH.D.
J. E. LEESON, D.V.M.
J. A. ARCHIBALD, D.V.M., M.V.SC.

A CONDITION pathologically and genetically indistinguishable from hemophilia A in man occurs in dogs.[1,3,5]

Two investigators[2] reported that they were able to breed and raise dogs affected with hemophilia A. These dogs have been useful in the study of hemophilia A and the nature of blood coagulation.

Knowledge of clotting mechanisms has advanced rapidly in man but has received little attention in veterinary medicine. We present the following simplified theory of blood coagulation, ignoring controversy over the terminology and the nature of various factors (fig. 1).

The defect in hemophilia A is considered to be a lack of factor VIII (antihemophilic globulin). Hemophilia B, the bleeding defect which is the subject of this report, is due to a lack of factor IX (Christmas factor).

Since domestic animals have been found to suffer from hemorrhagic syndromes similar to those observed in man, the veterinary practitioner should be aware of the nature of bleeding disorders. Most veterinarians engaged in practice or in research employing animals have observed variations in bleeding or blood coagulation and are aware of the difficulties that such an animal presents following blood loss from trauma or surgery.

From the Department of Medicine and Surgery (Archibald) and the Department of Physiological Sciences (Downie, Rowsell), Ontario Veterinary College, Guelph; Dr. Leeson is a practitioner in Toronto, Ont.; and Dr. Mustard is consultant, Department of Physiological Sciences, Ontario Veterinary College, and is associated with Sunnybrook Hospital, Department of Veterans' Affairs, Toronto, Ont.

Case Histories

A veterinary practitioner (J.E.L.) reported to the Small Animal Division of the Ontario Veterinary College his observations on 2 Cairn Terriers which bled excessively after minor surgery. Study of the coagulation defects led to the investigation of the kennel from which the dogs originated. Through the cooperation of the owner, the family tree (fig. 2) was determined, and 18 dogs were made available for further study.

The following is a history of the dogs, including the 2 mentioned, in which the bleeding tendency occurred. The numbers refer to the position of the dog in the family tree. All affected dogs to date have been males.

Dog 4.—A male, 2 years old, developed an extensive hematoma on its back as a result of a dog fight.

Dog 7.—A male, 1 year old, had its accessory digits removed at 4 months of age. The dog bled intermittently from the surgical site for 1 week despite the application of bandages and hemostatic agents.

Dog 8.—Following the removal of 2 temporary teeth this dog, a male, 1 year old, oozed blood continuously from the sockets for the next 7 days despite packing and suturing. Plucking of this dog resulted in a large ecchymotic swelling over the dorsal area. At a later date, hemorrhage believed due to trauma occurred in the anterior chamber of the left eye, producing a corneal opacity.

Dog 10.—A male, 5 months old, had excessive hemorrhage after its claws were clipped.

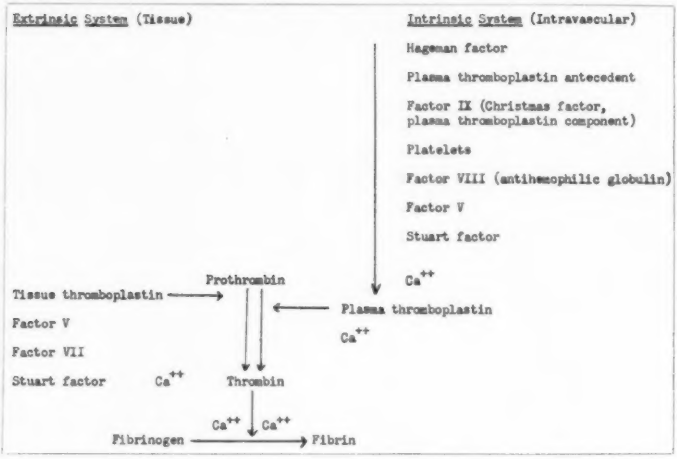

Fig. 1—Factors concerned in blood coagulation.

Family History

All the dogs were linebred Cairn Terriers. Those on which coagulation studies were carried out were given a number. Of the 18 available for study 4 (dogs 4, 7, 8, and 10) had abnormal clotting mechanisms, in addition to their history of a bleeding tendency. These 4 dogs were either sons or grandsons of the female listed as dog 1. None of the females in the litter of dog 1 had a history of the hemorrhagic tendency. All females tested had normal clotting mechanisms.

A female (dog 2) and a male (dog 3) littermate of dog 1 had no history of a bleeding tendency, nor did the offspring of dog 2. The clotting mechanisms were normal in dogs 2 and 3 and in the 8 offspring of dog 2. It is of interest that dog 6, an offspring of dog 2, sired 2 of the 4 litters in which affected dogs were found. When this dog was bred to dog 1, there were no affected pups.

No reliable information was available concerning the presence or absence of the bleeding tendency in the parents of dogs 1, 2, and 3. However, the sire of these dogs was from an imported line, and it had been reported from overseas sources that this breed of dogs contained members which were hemophilic.

After the initial study, dog 7 was bred to dog 1, resulting in a litter of 3 male pups and 1 female. Unfortunately, 1 male and the female died, before they could be studied, as the result of an infection contracted from the dam (dog 1). The female pup was small and was not considered viable when first observed. Of the 2 surviving male dogs, 1 is normal and the other has the clotting defect. Dog 7 recently fought with these 2 offspring. As a result, he suffered from a small penetrating tooth wound on the inner surface of his left upper lip. Bleeding occurred for several days. The oozing of blood ceased when 100 ml. of whole blood was transfused: the hemoglobin had dropped to 5 Gm./100 ml. The 2 pups subsequently fought and the pup with the bleeding tendency received a tooth wound in the external surface of the masseter muscles. A subcutaneous accumulation of blood developed which oozed for several

TABLE 1 — Coagulation Studies

Dog	Clotting time (min.)	Prothrombin time (sec.)	Russell's viper venom time (sec.)	Prothrombin consumption (sec.)	Platelets (No./cmm.)	Thromboplastin generation
Normal	8-14	8-13	7-12	18	150,000-400,000	Normal
4	+60	8.2	7.0	11.0	400,000	Serum abnormal
7	+60	9.8	10.2	11.5	320,000	Serum abnormal
8	+60	10.0	7.5	13.0	270,000	Serum abnormal
10	+60	8.5	8.5	9.0	500,000	Serum abnormal

days. The administration of 10 ml. of homologous antiserum stopped the bleeding, and 100 ml. of whole blood was transfused to correct a low hemoglobin level.

A second breeding of dog 7 to dog 1 has resulted in a litter of 3 pups, 2 males and 1 female. To date, coagulation studies have not been carried out on these pups because of their age.

Methods and Materials

Blood, collected from the cephalic vein of each dog, with a paraffiin-coated glass syringe and a 21-gauge silicone-coated stainless steel needle, was transferred to a variety of tubes on which various tests for the evidence of coagulation factors were carried out. The coagulation indexes included whole blood clotting time, prothrombin time, Russell's viper venom time, prothrombin consumption, calcium clotting time, platelet count, and thromboplastin generation tests. The tests were carried out using methods which have been described.[7-9]

Results of Coagulation Studies.—The results for some of these determinations are shown (table 1). The clotting times were prolonged in the 4 clinically affected males (dogs 4, 7, 8, and 10), as well as the 1 male offspring of dog 4 and dog 1. Clotting times were normal in the remaining dogs studied. The prothrombin time and Russell's viper venom time were normal in all dogs, as were the platelet counts.

The affected dogs had abnormal prothrombin consumption tests and abnormal thromboplastin generation tests. The defect in thromboplastin formation was found to be a serum factor which was corrected by the use of serum prepared from a normal dog or from a normal human being. The serum defect was corrected by the addition of equal parts of serum from human hemophilia A or plasma thromboplastin antecedent-deficient serum, but was not corrected by a serum from human beings with hemophilia B.

Radiobiological studies* on the platelets suggested, as in the human disease, that the biological half life of the platelets is similar in normal and affected dogs.

Detailed investigations and results of the coagulation studies will be reported in another publication.[10]

*Radiobiological studies on platelets were carried out by Dr. G. A. Robinson, Department of Physiological Sciences, Ontario Veterinary College, Guelph, detailed results to be reported.[10]

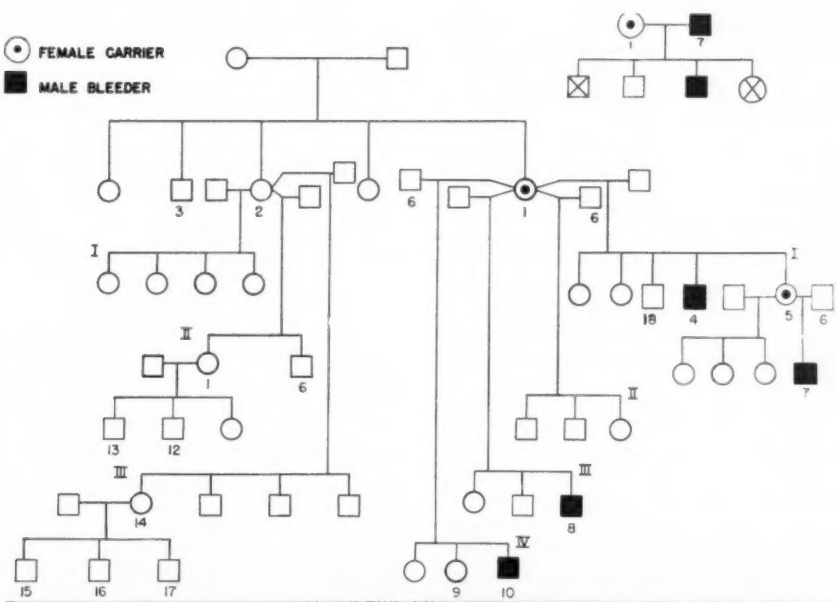

Fig. 2—Family history of dogs with hemophilia B. Arabic numbers indicate dogs available for study; Roman numerals indicate litter number whelped by females 1 and 2.

Discussion

The defect in these dogs resembled the human disorder known as hemophilia B or Christmas disease. The canine disease appeared to be inherited as a sex-linked recessive character. However, more extensive study is necessary to confirm this point. At the time of this report, the mode of inheritance appears similar to that in human hemophilia B (Christmas disease).

Veterinarians have recognized for some time that in highly inbred strains of dogs, it is not unusual to find some affected with genetically determined disorders. A familial canine chondrodystrophia foetalis (achondroplasia) has been described in 1 member of each of 4 consecutive litters of pedigreed Miniature Poodles.[4] The genetic and hereditary aspects of acetabular dysplasia in German Shepherd Dogs has been reviewed recently.[6,11]

The defect in blood coagulation is in the formation of plasma thromboplastin, and it is a serum factor. Comparison with the human disorder shows the defect to be diminished factor IX activity. Bleeding in affected dogs may be treated by intravenous administration of blood plasma or serum from normal dogs. Blood transfusions may be required to raise fallen hemoglobin levels.

It has been reported by breeders[12] that Cairn Terriers are susceptible to arthritis. Since arthritis is a common sequel to human hemophilia A and hemophilia B because of bleeding into the joint cavities, it is possible that some of the arthritis observed in Cairn Terriers may be due to this condition.

Aside from giving further evidence of bleeding abnormalities in dogs, investigation of Cairn Terriers and other breeds by veterinarians might make available a further source of these dogs for experimental work. The nature of blood coagulation has been assisted greatly by the study of hemophilia A in dogs.

In addition, the veterinary practitioner can strengthen the breed by having an awareness of bleeding abnormalities and by assisting kennel owners in eliminating such strains from their breeding stock. The failure of some breeders to recognize that genetically linked abnormalities may exist in their kennels may eventually doom a breed. The veterinarian should explain to kennel owners and breed associations that, while dogs affected with nonvisible abnormalities may win Best in Show ribbons, the perpetuation of this strain of dog will jeopardize the future popularity and acceptance of the breed. In addition, owners as well as kennel breeders can aid the study of human disorders by donating or providing affected animals to interested research groups.

Summary

A disease is reported in dogs of the Cairn Terrier breed, which is similar genetically and pathologically to hemophilia B or Christmas disease in man. It is characterized by a defective blood coagulation mechanism, due to a serum factor that affects thromboplastin formation. The disease appears to be inherited as a sexlinked recessive character, and in this study it was found to occur only in male dogs. Bleeding in affected dogs may be treated by the administration of blood plasma or serum from normal dogs.

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Effect of *Preanesthetic Medication with* Promazine and Promethazine on *Pentobarbital* Anesthesia *and Subsequent Hypothermia in the Cat*

Donald H. CLIFFORD, D.V.M., M.P.H., PH.D.

THE PURPOSE of the following experiments on hypothermia was to determine if moderate exposure to cold would have a greater effect on cats that were anesthetized with pentobarbital sodium alone or on cats that had been given promazine or promethazine prior to pentobarbital administration.

The fact that certain ataractic and anesthetic drugs may retard body metabolism at room temperature has been established.[10,12,19,22] Hypothermia, either local or general, may prevent shock[1,2,8] and allow surgical procedures in regions of the body or under circumstances where otherwise they would have been restricted.[3,16,24] Hypothermia lowers the basal metabolism and raises the pain threshold;[4,5,11,21] it may so prolong and intensify drug actions that they endanger life.[12,14,19]

Materials and Methods

All cats used in this series were examined and hospitalized for several days to establish that they were free from disease before they were anesthetized. Female cats were ovariohysterectomized and male cats were castrated to minimize sex differences. Preanesthetic agents (promazine* and promethazine*) were administered as previously outlined.[6] Pentobarbital sodium** was administered until the palpebral, digital, and ear-whisker reflexes were abolished. Following intravenous injection of pentobarbital, a 29-cm. Schaar centigrade thermometer was inserted approximately 16 cm. into the colon. The cats were then placed in an environmental temperature of 10 C. for a maximum of 4 hours. Colonic temperatures and respiratory and cardiac rates were measured at half-hour intervals. The temperature of the environment measured by a second thermometer increased an average of 2.8 C. during recording of the temperature and cardiac and respiratory rates.

During preliminary trials, it was observed that several cats which were subjected to low ambient temperatures recovered before the end of 4 hours. In order that definitive conclusions could be made, a system was devised by which the condition of each cat could be evaluated at the end of 4 hours, or at such time that it was necessary to remove the cat from the cold environment. The code was as follows: 0—no reflexes present; 1—palpebral, digital, or ear reflexes present; 2—shivering; 3—movement of head or limbs; 4—able to assume sternal recumbency. Some cats were removed from the cold environment in less than 4 hours since they regained their righting reflexes and it was feared that their movements might break the thermometers.

Since most cats were anesthetized more than once, at least 10 days were permitted to elapse between trials, which enabled the cats to fully recover. The effect of administration of 1 or more barbiturate drugs on subsequent administration of the same, or similar, drugs has been reviewed.[2] In order to compare the effect of pentobarbital used in previous anesthetic trials to the effect in present trials, 16 cats were reanesthetized with pentobarbital alone at the conclusion of the experiments (tables 1 and 2). Each of the first 7 groups contained 10 cats. The standard error of the mean was calculated.[20] Comparisons to the initial group of cats anesthetized with pentobarbital alone were made by Student's "t" test.[23]

Results and Discussion

The effects of doses of promazine (varying from 1 to 6 mg./lb. body weight) and promethazine on the anesthetic dose of pentobarbital and the colonic temperature, cardiac rate, respiratory rate, and return of reflexes of cats exposed to low ambient temperature (10 C.) for 4 hours were studied (tables 1 and 2). Additional comparisons with meperidine, chlorpromazine, trimeprazine, W 1527-63, and perphenazine were included in the original thesis.[7]

Anesthetic Dosage.—The effect of various preanesthetic doses of promazine and promethazine on the anesthetic dose of

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*Promazine (Sparine) and promethazine (Phenergan), Wyeth Laboratories, Philadelphia, Pa.

**Pentobarbital Sodium solution, Haver-Lockhart Laboratories, Kansas City, Mo.

TABLE 1—Statistical Comparison Between Groups of Cats Anesthetized with Pentobarbital Alone (Groups 1 and 8) and Groups of Cats Anesthetized with Pentobarbital Following Preanesthetic Administration of Promezine

Group No.	Promazine (mg./lb.)	Pentobarbital sodium anesthesia: Mean dose (mg./kg.)	S.E. ±	p (%)	Colonic temperature: Mean (C.)	S.E. ±	p (%)	Cardiac rate: Mean (rate/min.)	S.E. ±	p (%)	Respiratory rate: Mean (rate/min.)	S.E. ±	p (%)	Reflexes* (After 4 hr.): Mean reflex	S.E. ±	p (%)
1	0	33	6.4	.....	28.7	1.0	.....	95	13	.....	11	2.8	.....	1.5	.45	.....
2	1	27	2.0	10-20	30.2	1.1	30-40	93	10	>50	12	2.0	>50	1.3	.43	>50
3	2	25	2.7	5-10	32.1	1.0	2-5	118	10	10-20	17	2.0	5-10	1.7	.48	>50
4	3	25	1.4	< 1	31.8	0.8	< 1	111	9	20-30	14	1.4	30-40	1.7	.48	>50
5	4	26	1.8	2-5	28.2	1.6	>50	95	13	>50	12	2.0	>50	1.3	.45	>50
6	5	25	2.4	< 1	30.0	0.9	30-40	96	7	>50	13	1.6	>50	1.4	.31	>50
7	6	28	1.9	10-20	27.4	1.4	>50	82	11	>50	11	1.7	>50	1.2	.38	>50
8	0	35	4.5	30-40	30.7	1.9	10-20	92	11	>50	11	1.3	>50	1.6	.26	>50

After being anesthetized, cats were exposed to low ambient temperatures (10 C.) for 4 hours; colonic temperatures, cardiac and respiratory rates, and status of reflexes were measured every 30 minutes. All groups contained 10 cats except group 8, in which there were 16. Colonic temperatures, cardiac rates, and respiratory rates are the lowest mean values.

S.E.—Standard error of mean; p—probability.

*Reflex code: 0—no reflexes present; 1—palpebral, digital, or ear reflexes present; 2—shivering; 3—movement of head or limbs; 4—able to assume sternal recumbency.

pentobarbital was compared (tables 1 and 2). The maximum reduction in the dose of pentobarbital was attained following 2 mg./lb. of promazine and 4 mg./lb. of promethazine. It would, therefore, appear uneconomical to employ greater doses of these drugs to potentiate the anesthetic action of pentobarbital. Three and 5 mg./lb. of promazine produced a more significant (less than 1%) reduction in the anesthetic dose of pentobarbital. The 2 mg./lb. dosage of promazine was significant at the 5 to 10 per cent level, whereas 4 mg./lb. of promethazine was significant at the 2 to 5 per cent level. These variations are undoubtedly due to random sampling. The values obtained following reanesthetization of the cats with pentobarbital alone subsequent to the trials did not differ appreciably from these values obtained with pentobarbital alone at the start of the trials.

More pentobarbital was required when 6 mg./lb. of promazine or promethazine were administered than when smaller doses were given (tables 1 and 2). One would expect that decreasing doses of pentobarbital would be required as larger doses of preanesthetic were administered. The development of tolerance to pentobarbital, the plateau effect of increasing doses of pro-

TABLE 2—Statistical Comparison Between Groups of Cats Anesthetized with Pentobarbital Alone (Groups 1 and 8) and Groups of Cats Anesthetized with Pentobarbital Following Preanesthetic Administration of Promethazine

Group No.	Promazine (mg./lb.)	Pentobarbital sodium anesthesia: Mean dose (mg./kg.)	S.E. ±	p (%)	Colonic temperature: Mean (C.)	S.E. ±	p (%)	Cardiac rate: Mean (rate/min.)	S.E. ±	p (%)	Respiratory rate: Mean (rate/min.)	S.E. ±	p (%)	Reflexes* (after 4 hr.): Mean reflex	S.E. ±	p (%)
1	0	33	6.4	.....	28.7	1.0	.....	95	13	.....	11	2.8	>50	1.5	.45	.....
2	1	29	2.3	30-40	29.2	1.0	>50	97	12	>50	12	1.8	>50	1.6	.43	>50
3	2	28	1.9	5-10	29.3	0.5	>50	109	14	40-50	12	1.7	>50	2.0	.40	40-50
4	3	31	1.7	40-50	30.2	0.9	20-30	99	7	>50	11	1.0	>50	1.3	.31	>50
5	4	25	2.5	2-5	29.6	1.3	>50	118	14	20-30	12	1.4	>50	1.7	.40	>50
6	5	27	2.2	10-20	28.6	1.3	>50	99	9	>50	11	1.2	>50	1.7	.37	>50
7	6	30	1.4	10-20	30.4	0.9	20-30	97	10	>50	12	1.3	>50	1.5	.27	>50
8	0	35	4.5	30-40	30.7	1.9	10-20	92	11	>50	11	1.3	>50	1.6	.26	>50

After being anesthetized, cats were exposed to low ambient temperatures (10 C.) for 4 hours; colonic temperatures, cardiac and respiratory rates, and status of reflexes were measured every 30 minutes. All groups contained 10 cats except group 8, in which there were 16. Colonic temperatures, cardiac rates, and respiratory rates are the lowest mean values.

S.E.—Standard error of mean; p—probability.

*Reflex code: 0—no reflexes present; 1—palpebral digital, or ear reflexes present; 2—shivering; 3—movement of head or limbs; 4—able to assume sternal recumbency.

mazine and promethazine, and random variation in samples or central nervous system stimulation which is sometimes observed following the administration of phenothiazone derivatives may account for this apparent lack of consistency with the higher doses of preanesthetic agents.

No comparisons between pentobarbital potentiating action of promazine and promethazine are available in the cat, but some indirect evidence is available from investigations conducted with other species. In preanesthetic medication in man, it has been reported that chlorpromazine was twice as potent as promazine.[9] This same potency relationship of chlorpromazine as a preanesthetic drug used prior to barbiturate anesthesia has been reported in cats.[6] In man, higher dosages have been suggested for promethazine than for promazine.[9] Chlorpromazine has been observed to be 4 times more potent than promethazine in prolonging anesthesia in the mouse.[20] The pentobarbital potentiating effect of chlorpromazine has been shown to be greater than that of promethazine.[18] Thus, it may be assumed that the most effective preanesthetic dose of promethazine would be greater than that of promazine.

From work reported here, one could conclude that at least twice the dose of promethazine would be required (tables 1 and 2). As has been reported in man, ataractic drugs alone do not afford adequate preanesthetic medication, but must be combined with other agents such as meperidine, barbiturates, scopolamine, or morphine.[9,17] In other studies, it has been demonstrated that, when either promazine or promethazine is combined with meperidine, there is a significant (1% level) reduction in the anesthetic dose of pentobarbital required.[6,7]

Colonic Temperature.—In the preanesthetic dose range of 1 to 6 mg./lb. of promazine and promethazine, the colonic temperature dropped less when 2 mg./lb. of promazine and 3 mg./lb. of promethazine were administered prior to pentobarbital administration (tables 1 and 2). In cats anesthetized with pentobarbital alone, it is generally concluded that, if the body temperature remains near normal, the cats will recover from anesthesia much faster than when the temperature is below normal.

Cardiac Rate.—The highest cardiac rate was observed following 2 mg./lb. of promazine and 4 mg./lb. of promethazine (tables 1 and 2). When barbiturates are injected intravenously to effect anesthesia, they do not appear to be directly toxic to the myocardium or to alter significantly the cardiac rhythm or conduction, although large doses produce cardiac irregularities and even cardiac failure.[13] Hypothermia exerts a depressant effect on the heart,[4,5] and this is aggravated by pentobarbital.[15] The sparing effect of the preanesthetic drugs on the anesthetic dose of pentobarbital is undoubtedly a factor in the elevations of the cardiac rate. Although this trend is apparent, none of these values were significant (tables 1 and 2).

Respiratory Rate.—When promazine (2 mg./lb.) was administered, the respiratory rate was elevated, but this increase in the number of respirations was not significant (table 1). Promethazine did not appear to influence the respiratory rate (table 2). Death following administration of pentobarbital is usually due to depression of the respiratory center. Prior to death the respiratory rate is very slow, *i.e.*, 4 to 6 respirations per minute. A preanesthetic medicament that results in an elevation in the number of respirations helps to counteract the serious toxic action of pentobarbital. Respiratory rate, like cardiac rate, is depressed by hypothermia.[4,5] Some correlation between the colonic temperature, heart rate, and respiratory rate at the various doses of promazine and promethazine may be seen (tables 1 and 2).

Reflexes After Four Hours.—There was no real difference in the state of the reflexes after 4 hours (tables 1 and 2). Possibly, this was due to the depressant effect of the cold environment. In a previous study,[7] return of righting reflexes was not significant in cats that were given promazine, 2 mg./lb., prior to pentobarbital, spayed, and held at room temperature. Promethazine, 1 mg./lb., also had no effect. It is possible that if the reflexes had been evaluated at a later time, *i.e.* 8 hours after the cats were anesthetized, additional information might have been revealed.

Summary and Conclusion

The most satisfactory preanesthetic dose of promazine and promethazine prior to pentobarbital sodium anesthesia in cats was 2 mg./lb. and 4 mg./lb. respectively. In general, when cats were anesthetized with and without these drugs and were exposed to an environmental temperaure

of 10 C. for 4 hours, there was less drop in colonic temperaure and heart rate in cats that received preanesthetic medication. However, only promazine produced a significant elevation of the colonic temperature. This drug also produced an increase in the respiratory rate, but this increase was not significant. Neither preanesthetic drug changed the status of the reflexes at the end of 4 hours. These data suggest that hypothermia in cats subjected to a low ambient temperature (10 C.) is a product of the depth of anesthesia and duration of anesthesia rather than specific drug action. No significant degree of tolerance was developed by cats which were anesthetized with pentobarbital sodium alone at the beginning and the end of the experiment.

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Interdigital Cyst Therapy

If interdigital cysts occur in dogs repeatedly at the same place, surgery should be performed, but it is impractical if the cysts occur at different places. A simple treatment is to clip the hair between the toe pads every week. The affected foot should be soaked daily in hot water to which a tablespoon of salt has been added per pint. After drying with a towel, the skin should be painted with tincture of iodine.—*Gaines Dog Res., Fall, 1959.*

A Case Report—

Arrested Testicular Development

in the Horse

R. D. FRANDSON, M.S., D.V.M.
G. P. EPLING, M.S., D.V.M.
R. W. DAVIS, M.S., D.V.M.

ALTHOUGH cryptorchism in the horse is not uncommon, the anatomical variations herein reported are unique in the experience of the authors.

History

A light-breed colt, approximately 1 year old, was purchased through a sale ring for dissection in the laboratory of the Department of Anatomy, College of Veterinary Medicine at Colorado State University. The colt appeared normal except that no testicle could be palpated on the left side.

Following our routine procedure, the colt was anesthetized, exsanguinated, embalmed, and the arteries were injected with red latex. The colt was then used by students for dissection.

Macroscopic Findings

Testicle, internal genitalia, and associated structures appeared normal on the right.

The genitalia on the left side were definitely abnormal (fig. 1). No testicle could be seen or palpated. Instead, a peritoneal ligament extended from the area of the vaginal ring forward to attach to the dorsocaudal angle of the spleen (fig. 2).

Two small but well-defined arteries passed between the 2 layers of peritoneum from the aorta toward the vaginal ring. The gubernaculum testis appeared to be well developed.

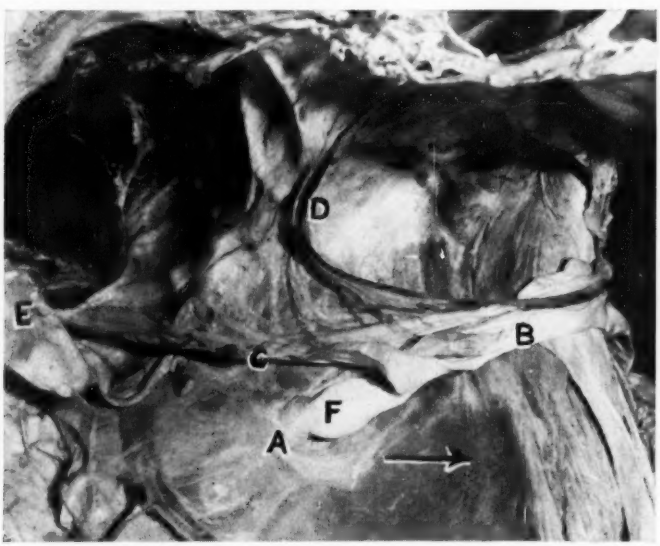


Fig. 1—View from right looking inside left flank of male horse cadaver (arrow points cranially) shows (A) vaginal ring, (B) peritoneal ligament extending from vaginal ring to dorsocaudal angle of spleen, (C) vas deferens, (D) spermatic vessels, (E) urinary bladder, and (F) gubernaculum testis.

From Colorado State University, Fort Collins, where Dr. Epling is professor, Dr. Frandson is associate professor, and Dr. Davis is professor and head of the Veterinary Anatomy Department.

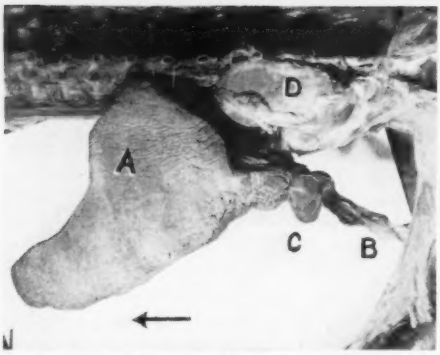


Fig. 2—View of horse cadaver from left side (arrow points cranially) shows (A) spleen, (B) continuation of peritoneal ligament extending from vaginal ring to dorsocaudal angle of spleen as in figure 1B, (C) site of adhesion between primitive testicle and spleen, and (D) kidney.

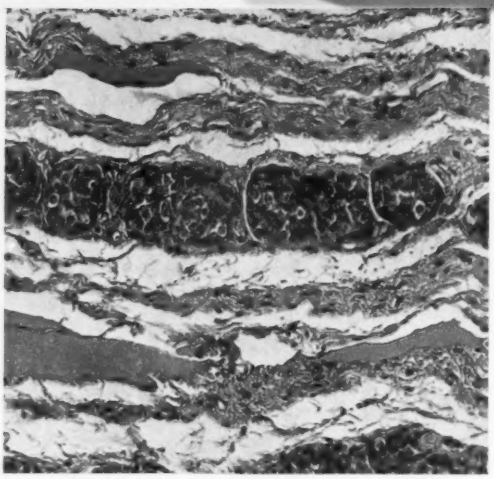


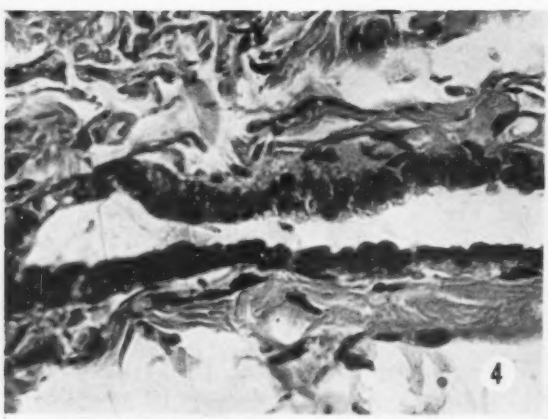
Fig. 3—Testis cord in areolar tissue near splenic capsule. x 120.

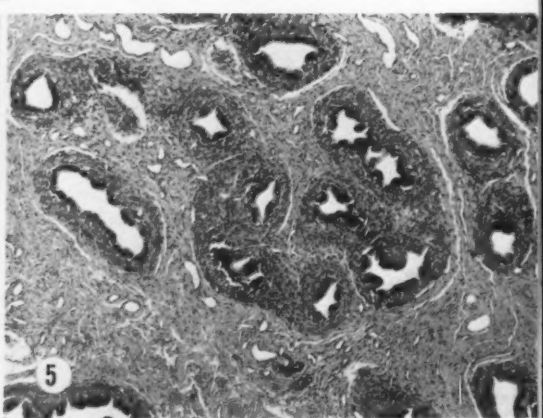
The vas deferens appeared normal as it passed caudally from the area of the vaginal ring to the prostatic urethra, but was difficult to trace cranially toward the spleen.

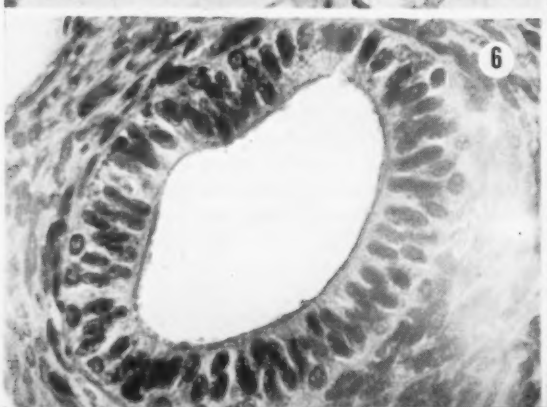
Microscopic Findings

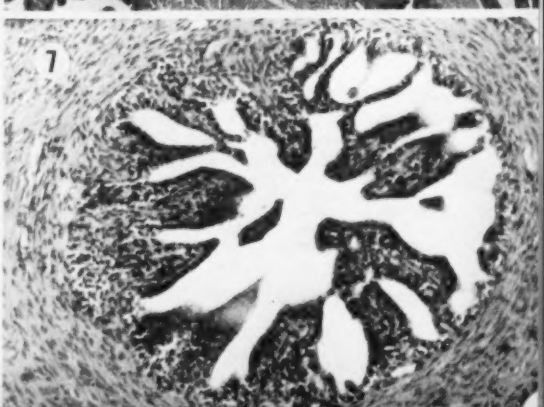
Immediately adjacent to the splenic capsule, a mass of loose vascular connective tissue (areolar) was observed which contained numerous testis cords like those one would normally expect to observe in the gonadal ridge of the developing male embryo. Some cells observed in these cords appeared to be differentiating into spermatogonia, but the majority appeared to be undifferentiated germinal epithelium. Some of the testis cords appeared to be developing a lumen, indicating their potential as convoluted seminiferous tubules (fig. 3). A few cells resembling the inter-

Fig. 4-7—Other structures found in the horse cadaver were (4) retained mesonephric tubule, x 385; (5) coils of epididymal duct, x 40; (6) epididymal duct, x 385; (7) vas deferens, x 100.









stitial cells of Leydig were observed in the vascular areolar stroma. Some retained mesonephric tubules were observed, apparently not yet fully modified into efferent ductules of the testis (fig. 4). All of these structures were included in an areolar stroma surrounded by a heavy dense irregular connective tissue capsule rich in smooth muscle fibers.

Further caudal, the connective tissue became more dense. This dense stroma contained the developing duct of the epididymis (fig. 5). The coils of the developing epididymal duct were loosely arranged, and showed heavy smooth muscle content in their walls. The smooth muscle became more voluminous as the duct was followed caudally. The lining epithelium was the type normally found in the adult epididymis (fig. 6).

The epididymal duct continued caudally as a vas deferens (fig. 7), which was coiled and invested by a heavy muscularis.

A heavy band of dense irregular connective tissue mixed with smooth muscle was observed extending from the vas deferens toward the scrotal region (fig. 1). This was the inguinal ligament of the mesonephros (the gubernaculum testis) destined in normal development to become the scrotal ligament.

Discussion

It appeared from gross and histologic evidence that the testicle was deficient in development and descent. Since it retained the characteristics and position of the gonadal ridge of the embryo, its fusion with the caudal aspect of the normally developed spleen was probably accidental. The spleen normally develops in the dorsal mesogastrium (greater omentum) and migrates to its left lateral adult location. This migration apparently brought the spleen in contact with the embryonic gonadal ridge, and an adhesion formed, thus anchoring the developing gonad firmly in position, reinforcing the diaphragmatic ligament of the mesonephros, and preventing the normal descent of the testicle.

This case is of particular interest when one considers the problem which would have confronted a surgeon had he been asked to perform a cryptorchidectomy on this colt. The structures which can usually be followed to the crytorchid testicle were present, *i.e.*, the vas deferens, gubernaculum testis, and vascular part of the spermatic cord. In this instance, however, none of these would have led to anything definitely palpable except the adhesion of testicular material to the spleen. Had this portion been removed surgically, the results might have been fatal.

Summary

A case of retarded testicular development in a colt is reported, in which adhesions developed between the spleen and embryonic gonadal ridge.

The Effect of Tranquilizers on Beef Cattle Performance

When weanling calves were given intramuscular injections of ethyl isobutrazine or a phenothiazine derivative referred to as SKF 5354A, only about half showed tranquilization, and this lasted less than 48 hours. Doses of 2.5 mg./kg. of body weight had greater effect than 1.5 mg./kg. The tranquilizer had no effect on weight gains of calves at weaning or the 21- to 30-day period following weaning. Addition of prochlorperazine to the feed of yearling steers did not improve rate of gain, feed efficiency, or carcass merit.

Small quantities of residual chlorpromazine were found in the fat, brain, heart, lung, and kidney of beef animals treated with the drug. Animals held for 72 hours after injection had no residual compound in any tissue. Lean muscle contained no residue, regardless of dose. It was concluded that tranquilizers had limited possibilities for increasing profit in meat animal production.—*Vet. Bull., 30, (Jan., 1960): Item 254.*

The Cause of Alimentary Toxemia in Chickens,

Toxic Fat

—*Its Effect on Swine Performance*

L. C. SCOTT, M.S.

IN 1957, a new disease entity of chickens occurred in many areas of the eastern United States. This condition has been adequately described.[3,4,7,9-12] Although commonly known as "water belly," various other descriptive terms such as alimentary toxemia,[9] hydropericardial disease,[4,7] toxopathic hepatitis,[11] endotheliosis,[12] chick edema syndrome,[8] and edema[1,2,13] have appeared in the literature.

The nature of field occurrences suggested the presence of a toxic principle in the feed. Preliminary investigations in many laboratories quickly eliminated common poultry diseases as causes. After considerable investigation, the unidentified toxic principle was found to be associated with certain shipments of fats which were added to poultry rations and investigations were initiated to determine the source of this toxic material. Although still unidentified, recent reports[1,2,5,6,8,13] added greatly to knowledge of the chemical characteristics of the toxic principle.

Many tons of feed had been prepared before the source of the trouble was recognized. Since this feed could not be fed to chickens, it was decided to ascertain if it could be safely utilized in swine feeding. It had been reported previously[9] that, "feed known to be carrying the toxic agent has been mixed with equal parts of ground corn and fed to fattening hogs for 60 days with no apparent harmful effects; the animals made excellent gains."

It was not known whether the pigs inactivated the toxin, were unaffected by it, eliminated it, or stored it unaltered in the body. It, therefore, seemed advisable to conduct further studies with higher levels of the toxic principle to determine, first, the effect on performance of swine, and second, to determine by biologic chick assay if the toxin was stored intact in the body when fed to swine. This paper reports the results of these studies.

Materials and Methods

Two comparable groups of 5 pigs each were confined in concrete-floored pens separated by a wooden partition. Each pen was provided with one 4-hole metal hog feeder. Water was provided in one water trough common to both pens. Equal shade and sleeping areas were provided for each group.

The feed used in the swine experiment contained approximately 15.8 per cent crude protein. Each 100 lb. of complete feed was composd of 16.5 lb. of a commercial 35 per cent protein supplement, 8.3 lb. dehulled solvent extracted soybean oil meal, 66.1 lb. ground yellow corn and 9.1 lb. animal fat. The fat used in the feed for one group of pigs was "normal" yellow grease of animal origin. The other group of pigs received fat known to contain the toxic agent.

After 29 days of feeding, 1 pig from the group fed toxic fat was selected and killed for special

Fig. 1—Pigs on left were fed 9.1 per cent toxic fat in the feed. Notice the marked difference in size and finish, compared with pigs the same age on right which were fed 9.1 per cent normal yellow grease in the feed for the same number of days.

Mr. Scott is associated with the Botkins Grain and Feed Co., Botkins, Ohio.

The author thanks Mr. Donald L. Manger, laboratory technician, Botkins Grain and Feed Co., for the care and management of the experimental animals, and Dr. Vance L. Sanger, veterinary pathologist, Ohio Agricultural Experiment Station, Wooster, for the necropsy, hematology, and histopathology which contributed much to the meaning of this paper.

studies, including a complete blood count. At necropsy, gross lesions were recorded. Representative samples of tissues including lung, liver, spleen, kidney, uterus, ovary, heart, aorta, esophagus, esophageal lymph node, psoas muscle, tongue, mesenteric lymph node, bladder, thymus, adrenal gland, stomach wall, duodenum, colon, hypothalamus, medulla, cerebellum, cerebrum, pituitary gland, bone marrow, and adrenal lymph node were stored in 10 per cent neutral formalin, embedded in paraffin, and sectioned at 5 μ. Hemotoxylin-eosin stain was used routinely and special stains were used where needed.

The remaining carcass of the pig was rendered in an open kettle in 2 separate portions — the fatty tissues, and the meat and bone tissues. The resulting fat samples were used in the chick-feeding study.

In the chick study, 48 straight-run, day-old Vantress x Pilch White Rock broiler chicks were allotted to 3 groups of 16 birds each and housed in electrically heated batteries. Feeding and management practices were equal for each group. A corn-soybean type diet containing 4 per cent of normal yellow grease of animal origin or one of the fat samples rendered from the pig was fed *ad libitum* to each group for 4 weeks. At the conclusion of this feeding trial, weights and feed efficiencies were recorded. Necropsies and gross observations were made on 5 birds from each group.

Results and Discussion

The pig-feeding trial began on May 26, 1958, and was concluded July 24, 1958—a period of 59 days. From the outset of the feeding period, the pigs fed the toxic fat showed a marked disinclination to consume a normal amount of feed. Although these pigs appeared active and in good general health, gains and feed utilization were drastically curtailed. Pigs consuming feed containing normal yellow grease appeared normal in all respects, made excellent gains, and utilized feed efficiently (fig. 1). Growth and feed utilization data are shown (table 1).

At the conclusion of the feeding trial, the 4 remaining pigs fed toxic fat were placed on the feed containing yellow grease for 75 days before being marketed. Feed consumption increased dramatically. These pigs were marketed at an average weight of 221 lb. and gains were a substantial 1.71 lb. per day for this period.

Throughout the chick study, no clinical signs of alimentary toxemia were observed in any group. No differences were observed in performance of these chicks (table 2). No gross lesions were observed at necropsy.

In a preliminary feeding trial in pigs,[9] chicken feed carrying the toxic fat at a 2.5 per cent level was mixed with equal parts of yellow corn. Pigs fed this mixture ate well and made good gains. However, in contrast to these results, pigs fed a ration containing 9.1 per cent of the toxic fat had poor appetites and their gains were noticeably retarded. Apparently the lower concentration of fat, plus the 50 per cent dilution of the original feed, reduced the unpalatability of the feed to such a point that it was not refused and the small amount of the toxic fat did not affect gains. Very likely part of the slower gains in the 5 pigs in this second feeding trial was a result of inadequate feed intake but it also seems probable that the toxic principle depressed growth rate to some degree. From these limited data, it can be concluded that chicken feed carrying the toxic fat at a 2.5 per cent level can be mixed with equal parts of yellow corn and fed to hogs with no ill effects. However, the toxic fat used in this experiment cannot be mixed at a 9.1 per cent level in regular hog feed without seriously depressing appetite and weight gains although the hogs remain active and healthy.

Also, the chick feeding trials indicated that the toxic principle was not stored in the fat of the pig which consumed toxic principle through the ration. This is in contrast to findings reported by other in-

TABLE 1—Performance of Pigs Fed Yellow Grease or Toxic Fat

	Yellow grease	Toxic fat
No. pigs started	5	5
No. pigs finishing test	5	4
Av. starting weight (lb.)	64	58
Total No. pig days	295	236
Total gain (lb.)	610	170*
Av. daily gain (lb.)	2.07	0.72*
Feed per 100 lb. gain	278	419*
Av. final weight (lb.)	186.0	92.5

*Includes gains and feed consumed by pig killed for special studies.

TABLE 2—Performance of Chicks Fed Yellow Grease and Fat from Pig Fed Known Toxic Fat

Ration variable	Started	Mortality	Av. 4-week weight (Gm.)	Feed gain
Yellow grease (4%)	16	1	419	1.80
Rendered fat from meat and bone (4%)	16	1	414	1.82
Rendered fat from fatty tissue (4%)	16	1	417	1.81

vestigators,[5] who found the toxic principle in the flesh of chickens in significant quantities after they were fed the toxic fat. However, these chicks may have consumed proportionately more toxic principle than the pig which was used here.

It was not determined what disposition the pig's body made of this toxic principle.

Necropsy, Gross and Microscopic Findings

The pig selected for detailed study weighed 90 lb. when killed. Clinically, it was active and in good health. A single hemogram just before slaughter revealed 7,540,000 erythrocytes, 12,500 leukocytes, 14 Gm. hemoglobin, 47 per cent segmented neutrophils, 50 per cent lymphocytes, 2 per cent monocytes, and 1 per cent eosinophils.

The only gross lesion was a small consolidated area of pneumonia in one lung with lungworms in and adjacent to the lesion.

Microscopically, the lung lesion showed fibrosis, neutrophilic infiltration, thickened alveolar walls, and lymphoid hyperplasia around some small bronchioles. Cross sections of lungworms were seen in the tissue. Peribronchial pneumonia was apparent also. There were no microscopic lesions in the other tissues studied including 5 different sections from the central nervous system.

Summary and Conclusions

Two comparable groups of 5 pigs each were fed rations containing 15.8 per cent protein and 9.1 per cent of either yellow grease or a fat known to contain the toxic agent associated with field outbreaks of alimentary toxemia, an edematous condition in chickens. The pigs fed yellow grease made excellent gains and converted feed efficiently. Pigs fed the toxic fat had marked inappetence and consequent poor gains and feed utilization. However, these pigs appeared healthy and active.

One pig selected from the group fed toxic fat was slaughtered and examined grossly and histopathologically. The remainder of the carcass was rendered to obtain fat for chick studies.

At the conclusion of the 59-day test period, the 4 remaining pigs were given feed in which the yellow grease was substituted for toxic-fat. Subsequent gains for 75 days were 1.71 lb. per head daily, thus indicating no permanent damage had been sustained by these pigs. Final sale weight was an average of 221 lb. per head.

Also, chick growth studies using a ration containing the 4 per cent fat rendered from 1 of the pigs in the group fed toxic fat indicated no appreciable amount of the toxic agent was stored in the fat or meaty tissues of the pig. When these chicks were necropsied, no gross lesions were present which could be associated with the lesions found in chickens that had been fed a toxic fat. The performance and appearance of the chicks were normal in all respects.

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A Survey of the Species of

Coccidia in Chickens

in Maine

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ONE OF THE greatest deterrents to successful poultry raising is a constant disease problem, one of the most troublesome of which is coccidiosis. During the previous decade, coccidiostats have been used extensively but, although partially successful, they have not been considered the solution to the coccidiosis problem.

The poultry industry (poultrymen, processors, feed companies, state and university officials) in Maine, concerned about the coccidiosis problem, made a survey of the state to determine which of the following species of Eimeria were present; *E. maxima, E. brunetti, E. acervulina, and E. hagani.* It was conceded that *E. tenella* and *E. necatrix* were widespread.

Few such surveys have been reported. One worker,[2] in 1937-1938 in New York State, examined 39 chickens from 33 farms and reported the incidence to be *Eimeria praecox*, 33 per cent; *E. maxima*, 28 per cent; *E. necatrix*, 38 per cent; and *E. tenella*, 23 per cent.

Two research workers,[1] (1950) in a general discussion of the distribution of the species, concluded that *E. maxima* and *E. acervulina* were widely distributed in the United States. They postulated that *E. tenella* and *E. necatrix* might be limited in certain areas; *E. mitis* and *E. praecox* were widely scattered; and *E. brunetti* and *E. hagani* were reported only from New York State.

Another worker,[4] who studied the parasitic fauna of 207 chickens on 2 farms in central Iowa, found *E. acervulina, E. brunetti, E. maxima, E. mitis, E. necatrix,* and *E. tenella* on one farm and all but *E. necatrix* on the second. Seven other species of intestinal parasites were also recorded.

Another worker[3] stated that all 8 species of Eimeria parasitic to chickens were present in Alabama. *Eimeria tenella* and *E. necatrix* were listed as common; *E. praecox, E. mitis,* and *E. acervulina* as less common; and the other 3 species as rare parasites.

This survey was based on the assumptions that: (1) Immunity is species specific. (2) All chickens are susceptible to infection with any given species until such time as they have become infected with that species and have survived. Subclinical or low-grade infections will induce demonstrable immunity to subsequent exposure to the same species but not to others. (3) The demonstration of resistance to a challenge inoculation of a pure culture of viable oocysts is valid presumptive evidence that the resistant bird has been previously infected with the test species. (4) Certain species can be reliably distinguished one from the other only on the basis of immunity. For these reasons, the most practical and reliable procedure for conducting a survey of enzootic species of coccidia is to determine the presence and frequency of resistance to challenge infection by the various recognized species.

An immunologic survey can be carried out by inoculation of proved infective oocyst suspension into mature farm birds collected on the basis of random sampling. The various species are characterized by differences in incubation periods, and by differences in elective localization of lesions. For this reason, the survey procedure was simplified by using a challenge

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The Wirthmore Feed Co., Waltham, Mass., supplied the feed used in the experiment, and with Beacon Feed Co., Samuel Lipman and Co. Feeds, Unity Feed Co., Purina Feed Co., and Eastern States Feed Co., supplied service personnel to pick up and transport the birds and assist at time of necropsy. The Fort Halifax Packing Co., Winslow, Maine, supplied the building and batteries for the experiment.

The following Maine veterinarians assisted: Drs. K. H. Eskelund, Waterville; J. L. Rountree, Augusta; J. Valentine, Belfast; A. Zarkower and R. L. Witter, Orono; and W. P. Williams, Atlanta, Ga.

TABLE 1—Inoculum and Number of Oocysts Given Control Groups

Group	Eimeria species	Oocyst challenge* dose/bird	Examined after challenge (days)
1	E. acervulina	2,000,000	6
	E. hagani	2,000,000	
2	E. maxima	400,000	6
	E. tenella	200,000	
3	E. brunetti	200,000	7
4	E. praecox	1,000,000	5

*Challenge inoculum supplied by Sterwin-Chemicals Inc., New York.

inoculum consisting of a mixture of 2 species which had the same incubation period but which differed in the distribution of lesions in the digestive tract.

In the study reported here, we attempted to survey as many farm flocks from the principal poultry-producing counties of Maine as was practical. The several flocks represented different breeds, feeds, and feeding and management practices.

TABLE 2—Percentage of Chickens Which Were Immune to Different Species of Eimeria

Group	Species	Immune (%)	Susceptible (%)
1	E. acervulina	84	16
	E. hagani		
2	E. maxima	91	9
	E. tenella		
3	E. brunetti	77	23
4	E. praecox	80	20

Materials and Methods

Mature hens, 30 weeks of age or older, were selected from 16 farms in 7 counties (Cumberland, Kennebec, Somerset, Knox, Lincoln, Waldo, and Androscoggin) which have the greatest population of laying hens (fig. 1). A total of 400 apparently healthy birds, 25 from each farm, were brought to one central location, "weighed in," leg-banded, and allotted to groups of 5 per cage. Four groups of birds from each farm were placed in 4 batteries for challenge, and the 5 birds comprising the fifth group were placed in a separate battery to serve as negative controls for necropsy and weight change studies. Complete data regarding owner, location, flock, size, age, feed used, coccidiostat used and how long, feeding management,* and disease history were obtained.

The birds were placed in clean, disinfected, 16-compartment, wire-floored cages and the papers were changed daily. No infectious disease signs were observed during the experiment. Birds were kept on nonmedicated feed 7 to 15 days prior to challenge. Only one challenge inoculum was used per cage.

A measured amount of the coccidial suspension was placed, with a glass pipette, directly into the crops of 2 groups of 25** susceptible 10-week-old birds.

Known susceptible control chickens were inoculated first and then the 5 controls

*"Full feed" indicated continuous feeding. "Controlled" feeding indicated that the birds were given a recorded (controlled) amount of feed each day. "Semicontrolled" feeding meant that the birds were given an unmeasured amount of feed until a given time each day (i.e., 3 p.m.), then did not receive any more until the following day.

**Twenty-five White Rock broilers were provided by the Wirthmore Feed Co., Waltham, Mass., and 25 White Leghorns from the University of Maine, Orono.

TABLE 3—Weight Changes, Gross and Microscopic Studies of Chickens (from 16 Maine Farms) Which Were Inoculated with E. acervulina and E. hagani

County	Farm No.	Feeding program	Arrival weight* (lb.)	Challenge weight* (lb.)	End weight* (lb.)	Species	
						E. acervulina immune	E. hagani suscept.
Waldo	1	Full	22.0	23.5	23.0	4	1
Cumberland	2	Semicontrol	25.5	26.5	27.5	4	1
Androscoggin	3	Controlled	22.0	27.5	27.5	4	1
Waldo	4	Full	29.0	29.5	29.0	2	3
Cumberland	5	Full	27.0	29.5	29.5	5	0
Lincoln	6	Full	32.0	31.5	31.5	5	0
Waldo	7	Controlled	29.0	30.5	30.5	5	0
Kennebec	8	Full	31.0	30.0	29.5	4	1
Somerset	9	Full	40.0	39.5	39.0	4	1
Knox	10	Full	25.0	25.5	25.75	4	1
Kennebec	11	Full	38.0	36.5	36.0	5	0
Kennebec	12	Full	30.0	31.5	32.0	4	1
Cumberland	13	Full	33.5	33.5	34.0	5	0
Androscoggin	14	Full	37.5	37.5	38.5	4	1
Kennebec	15	Controlled	27.0	28.0	28.0	5	0
Waldo	16	Controlled	27.5	31.0	31.0	3	2
Totals 7	16					84%	16%

*The total weight of the 5 birds in each group.

TABLE 4—Weight Changes, Gross and Microscopic Studies of Chickens (from 16 Maine Farms) Which Were Inoculated with E. maxima Oocysts.

County	Farm No.	Feeding program	Arrival weight* (lb.)	Challenge weight* (lb.)	End weight* (lb.)	Species E. maxima immune	Species E. maxima suscept.
Waldo	1	Full	20.0	21.5	21.5	5	0
Cumberland	2	Semicontrol	24.5	27.5	27.5	4	1
Androscoggin	3	Controlled	23.0	28.5	29.5	5	0
Waldo	4	Full	26.0	26.5	26.5	5	0
Cumberland	5	Full	28.0	30.0	30.0	5	0
Lincoln	6	Full	35.0	36.0	37.0	4	1
Waldo	7	Controlled	31.0	30.5	31.5	3	2
Kennebec	8	Full	32.0	31.5	32.0	4	1
Somerset	9	Full	38.0	37.0	36.5	3	2
Knox	10	Full	28.0	29.5	30.0	5	0
Kennebec	11	Full	32.0	34.5	35.0	5	0
Kennebec	12	Full	36.0	36.0	36.5	5	0
Cumberland	13	Full	31.5	34.0	35.0	5	0
Androscoggin	14	Full	39.5	40.5	39.5	5	0
Kennebec	15	Controlled	24.5	23.5	23.75	5	0
Waldo	16	Controlled	28.5	30.0	30.0	5	0
Totals 7	16					91%	9%

*The total weight of the 5 birds in each group.

from each of the 16 farms were inoculated. The 4 challenge inoculums and number of oocysts in each dose are shown (table 1).

The birds were weighed at the time of challenge and just prior to necropsy.

The presence or absence of immunity was established at the time of necropsy. After inoculation, the infected controls were examined at necropsy. Extent and nature of gross lesions were observed and the presence of the characteristic oocysts were confirmed by microscopic examination. The farm-reared controls were then necropsied and their immunity status determined before proceeding to the next group. At the time of examination, representative tissues from each chicken were stored in 10 per cent formol-saline solution for histologic studies.

Results

The evaluation of immunity was made by the following criteria: (1) lesion score —gross pathologic findings and microscopic demonstration of oocysts; (2) weight change from date of challenge to necropsy.

TABLE 5—Weight Changes, Gross and Microscopic Studies of Chicken (from 16 Maine Farms) Inoculated with E. praecox Oocysts

County	Farm No.	Feeding program	Arrival weight* (lb.)	Challenge weight* (lb.)	End weight* (lb.)	Species E praecox immune	Species E praecox suscept.
Waldo	1	Full	21.0	22.5	22.25	4	1
Cumberland	2	Semicontrol	26.5	29.5	30.0	5	0
Androscoggin	3	Controlled	27.0	24.0(1 died)	24.0	2	2
Waldo	4	Full	27.0	26.5	26.75	5	0
Cumberland	5	Full	20.0	30.0	31.5	3	2
Lincoln	6	Full	31.0	23.0(1 died)	23.5	4	0
Waldo	7	Controlled	29.0	29.0	29.5	4	1
Kennebec	8	Full	31.5	32.0	31.0	0	5
Somerset	9	Full	38.0	37.5	37.0	3	2
Knox	10	Full	29.0	30.5	30.25	5	0
Kennebec	11	Full	34.0	34.5	35.5	5	0
Kennebec	12	Full	30.0	29.5	29.5	4	1
Cumberland	13	Full	33.0	34.5	35.0	5	0
Androscoggin	14	Full	37.5	38.5	38.5	4	1
Kennebec	15	Controlled	26.5	28.0	30.0	4	1
Waldo	16	Controlled	26.0	28.0	29.25	5	0
Totals 7	16					80%	20%

*The total weight of the 5 birds in each group.

TABLE 6—Weight Changes, Gross and Microscopic Studies of Chickens (from 16 Maine Farms) Inoculated with E. brunetti Oocysts

County	Farm No.	Feeding program	Arrival weight* (lb.)	Challenge weight* (lb.)	End weight* (lb.)	Species	
						E. brunetti	
						immune	suscept.
Waldo	1	Full	22.0	23.5	24.0	5	0
Cumberland	2	Semicontrol	28.5	30.0	31.0	4	1
Androscoggin	3	Controlled	23.0	26.5	26.0	5	0
Waldo	4	Full	29.0	26.0	25.5	2	3
Cumberland	5	Full	30.0	32.5	31.5	2	3
Lincoln	6	Full	33.0	32.0	33.5	5	0
Waldo	7	Controlled	26.5	29.0	29.5	5	0
Kennebec	8	Full	29.5	28.0	28.0	4	1
Somerset	9	Full	38.0	37.0	36.0	5	0
Knox	10	Full	28.0	30.0	29.0	1	4
Kennebec	11	Full	36.0	34.5	34.5	5	0
Kennebec	12	Full	30.0	30.5	31.0	5	0
Cumberland	13	Full	30.0	32.0	31.0	5	0
Androscoggin	14	Full	38.0	37.5	37.5	5	0
Kennebec	15	Controlled	21.5	23.5	21.5	1	4
Waldo	16	Controlled	25.5	26.0	26.0	3	2
Totals 7	16					77%	23%

*The total weight of the 5 birds in each group.

The results of the experiment, using an inoculum containing *E. acervulina* and *E. hagani*, are indicated (table 3). Although birds in every flock had some degree of immunity to this challenge, those from farms 4 and 16 had the least.

Although 9 per cent of the birds were susceptible to *E. maxima* (table 4), all chickens challenged from 11 of the 16 farms were completely immune to this species. Blood was not observed grossly in any of the ceca studied. Under the conditions of the experiment, it was impossible to differentiate *E. maxima* and *E. tenella* oocysts.

Twenty per cent of the birds whose immunity was challenged were susceptible to *E. praecox* (table 5). The 5 control birds from farm 8 were all susceptible. Flocks on 6 farms were completely immune, but on most farms 1 or more birds were susceptible to these coccidia.

Flocks on 9 farms were completely immune to *E. brunetti*, whereas on 4 farms, more than 50 per cent of the chickens were susceptible (table 6). Of all the birds challenged, 23 per cent were susceptible to these coccidia.

Although weight differences were recorded, they were not conclusive, as birds in some lots increased in weight whereas

TABLE 7—Weight Changes in Control Chickens

County	Farm (No.)	Feeding program	Weight when received* (lb.)	Weight before challenge of test birds (lb.)	End weight
Waldo	1	Full	22.25	20.0	20.5**
Cumberland	2	Semicontrol	22.0	21.5	24.0
Androscoggin	3	Controlled	25.0	23.0	23.0†
Waldo	4	Full	40.0	40.0	40.0
Cumberland	5	Full	30.0	26.0	25.5‡
Lincoln	6	Full	26.0	20.0	18.5§
Waldo	7	Controlled	27.5	29.0	29.5
Kennebec	8	Full	17.5	16.5	16.0
Somerset	9	Full	33.5	29.5	25.0#
Knox	10	Full	25.0	28.5	28.0
Kennebec	11	Full	35.5	35.5	35.0
Kennebec	12	Full	34.0	32.5	33.0
Cumberland	13	Full	33.0	32.5	32.0
Androscoggin	14	Full	22.5	23.0	23.0
Kennebec	15	Controlled	23.5	24.0	24.0
Waldo	16	Controlled	26.5	28.0	29.5

*The total weight of the 5 birds in each group; **bird 19025, 4.0 lb., was placed in the group given E. acervulina from farm 1; †bird 1919078, 5.5 lb., died June 26, 1959; ‡bird 19133, 4.0 lb., placed in group given E. maxima from farm 5; §bird 19161, 4.5 lb., died July 3, 1959; #bird 19230 died July 16, 1959.

those in other lots decreased. It did appear that, generally, an increase or no change occurred when all 5 birds were immune to challenge.

All birds on semi- or controlled feeding programs increased in weight when placed on full feed (table 7).

All the infected susceptible control birds developed typical disease lesions. with many oocysts present.

Susceptible challenged controls had all the clinical signs typical of the species studied. Five flocks, farms 3, 4, 5, 15, and 16 of the *E. brunetti* challenged groups, showed soft, wet droppings. Of the *E. acervulina* group, those from farms 10 and 11 had slight diarrhea and those from farm 6 marked diarrhea. In the *E. maxima* group, diarrhea and some blood was observed in farm 7, and diarrhea in farm 8.

When the immunity of susceptible controls was challenged with *E. praecox*, huddling, poor appetite, and slight diarrhea occurred. On necropsy of the birds, enteritis, ballooning of the intestines, and numerous oocysts were observed.

Discussion

From these studies, it would appear that all the species of coccidiosis studied were widespread; however, the fact that birds on an occasional farm are susceptible, might indicate that some farms are not yet contaminated. Naturally, in any study of immunity the degree of exposure to infection is critical. This study reemphasizes that "immunity is a relative thing." It is possible that a stronger challenge dose might have revealed fewer birds with a greater immunity, or a lesser challenge might have revealed more immune birds. The *E. praecox* challenge indicated that this species may be more pathogenic than has been reported in the past. Certainly this area of study is open to further investigation.

This study can be considered to be representative of environment, feeding programs, management, and breeds of chickens in Maine.

Summary

1) A study of *Eimeria acervulina, Eimeria hagani, Eimeria maxima, Eimeria tenella, Eimeria praecox,* and *Eimeria brunetti* infections in adult fowl from Maine indicated these species to be widespread in 7 counties.

2) There was considerable variation in the number of immune chickens in the different flocks.

3) The actual percentage of chickens which had become immune to the different species is shown (table 2).

4) From only one farm were all 5 controls susceptible to *E. praecox*.

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Teraphthalic Acid Tested for Lamb Urolithiasis

Teraphthalic acid was included in the diet of lambs at a level of 0.3 per cent in an attempt to evaluate its potentiating ability on oxytetracycline and the resulting effect on the development of urolithiasis. Of 96 lambs given teraphthalic acid, 1 had calculi in the urinary tract as compared with 9 of 122 lambs that did not receive teraphthalic acid. Occurrence of urinary calculi was too limited to evaluate effectively the action of teraphthalic acid when fed alone or in combination with antibiotics. Teraphthalic acid may be included in the diet of fattening lambs at 0.3 per cent without significantly affecting feed consumption or rate of gain.—*J. Anim. Sci., 18, (1959): 1501.*

Our Medical and Agricultural Relationships

R. E. NICHOLS, D.V.M., P.H.D., D.V.SC.

MAN HAS benefitted greatly from agriculture, medicine, and veterinary medicine. Healthy expansion of each will continue to offer him better health and food; however, within these fields there are specialty groups which are reluctant to change to grasp new opportunities.

One of the limitations of specialization is the failure of one group to recognize that man needs not only its contributions but those of all other specialties as well. This has led to more competition than cooperation between important facets of our professions. Large, well-financed groups tend to "steamroller" over smaller ones. This type of activity eventually restricts the benefits that man has a right to expect from his professions.

Perhaps we need to expose our future specialists during their training period to information about all the specialties in veterinary medicine. Should not the selection of a specialty be based primarily on the individual's clear idea of its relative position in the profession rather than on the potential income it seems to offer?

Interprofessional understanding can be strengthened not by criticizing other professional groups, but by supporting them. We also need to encourage the kind of support from agriculture and medicine which comes from their understanding of veterinary medicine—not their ignorance of it.

It has been suggested that the teaching of human and veterinary medicine be combined. Certainly some phases of technical information are common and may be taught jointly with medical students. But what about systems of husbandry, farm animal nutrition, and market values of livestock? Would the medical faculty understand these basic and economic problems, or would it slant its emphasis toward public health alone?

On the other hand, there are those in agriculture who still feel that emphasis in veterinary education should be limited to their special problems. Certainly the veterinary curriculum must include livestock breeding, feeding, and marketing. But would the agricultural college fully appreciate the necessity of training veterinarians in the public health aspects of animal disease control?

Thus it would seem that there is need of liaison with both the medical and agricultural schools, neither of which is staffed or equipped to teach the kind of veterinary medicine which the newer, more concentrated systems of animal production will require. They can both help.

Regardless of the family farm and other social implications, animal production is traveling rapidly toward corporate-size units. It is now seldom possible to treat large numbers of diseased food animals profitably. This emphasizes control of animal diseases at the production level. It deemphasizes the sorting out of diseased food after it has reached avenues of distribution, where dissemination of disease can be more rapid.

Control of disease at the production level could reduce the sale and movement of diseased animals. Perhaps some day it will be illegal to accept diseased animals at the same places where healthy food is processed. Perhaps some day a processor will be allowed to purchase and ship only those animals which come from certified healthy herds or which have been examined on the farm and declared healthy by a qualified veterinarian. The economic incentives for raising healthy stock may require greater emphasis on contract disease prevention and closer association of the veterinarian with actual operations of animal producing units. He will be expected to justify economically his recommendations and practices of disease control. Will he be prepared to meet this trend? Will he be prepared medically *and* agriculturally?

Dr. Nichols is a professor of veterinary science at the University of Wisconsin, Madison.

Editorial

Is Mass Use of Tranquilizers in Beef Cattle Justified?

Tranquilizers have received a great deal of attention in the field of veterinary medicine in recent years, and many valuable uses have been found for them. Along with the valuable uses discovered, a number of "suggested" or "possible" uses have been brought to the attention of veterinarians and livestock men, especially involving injectable tranquilizers. These suggested uses included tranquilization of beef cattle to reduce shrinkage associated with loading, unloading, and transport, reduce bruising and injury incurred in connection with transit, improve feed consumption and weight gains in feeder cattle, reduce incidence of shipping fever, and facilitate adjustment of calves to feedlot conditions.

However, many hopeful veterinarians and their clients have been disappointed with the results they obtained. Benefits derived from use of injectable tranquilizers have frequently been offset by the added handling time and expense necessitated by restraint of cattle in order to administer the drug.[4,5,14] Benefits also have been minimized or eliminated by the cost of the tranquilizer itself.[4,5,14] On occasions, cattle tranquilized before shipment to packing plants developed sizable necrotic areas at the injection site. Pounds of flesh had to be trimmed from the carcass to remove these areas, and carcasses were downgraded.[4] During shipment, some cattle were bruised more than normally and even crippled because they were unable to protect themselves or because they lay down in cars and were stepped on by standing cattle.[1,4] Some cattle were so depressed by tranquilizers that loading and unloading became an expensive and laborious process.[5,14] Furthermore, carefully conducted trials indicated that tranquilizers were of little or no benefit in reducing shrinkage and occasionally increased shrinkage in cattle during transit to feedlots or to slaughter.[1,4-6,10,11,14]

In a trial conducted by the Ohio Agricultural Experiment Station, in which 180 feeder calves were involved, shrinkage in tranquilized calves was 9.3 per cent, whereas in untreated calves it was only 9.1 per cent. In tranquilized fat cattle and feeder calves shipped by truck for short distances shrinkage was less than in controls but the weight saving was not enough to compensate for the cost of the drug and its administration.[8] Similar results have been reported by other investigators.[5,6,11,15] Ability of tranquilizers to increase weight gains and feed consumption in feedlot cattle has not been proved.[1,2,5,10]

Feed additive tranquilizers so far have been shown to have little more value than injectable types.[1,3,8,9,12,13,15]

The incidence of shipping fever generally has not been reduced by use of tranquilizers. According to one investigator who conducted experiments on 180 cattle, incidence of shipping fever was slightly greater in tranquilized cattle than in control cattle.[7] Another investigator stated, "In connection with our feedlot studies in Illinois, we followed some lots where tranquilizing drugs had been used prior to shipment. Shipping fever appeared to be just as frequent and as severe in those lots as in those not treated."[5] On the other hand, a third investigator reported that over a 2-year period 573 range calves were given 3 different tranquilizers prior to shipment to feedlots, with favorable results. Tranquilized cattle had a shipping fever incidence during the first 3 weeks in feedlots of 4.0 per cent; other prophylactically treated calves, an incidence of 6.1 per cent; while untreated control calves had an incidence of 7.1 per cent. Although these figures indicate that tranquilizers had a favorable influence on incidence of shipping fever, the investigator commented that "Careful handling during transportation, loading, and unloading, rest upon arrival at the feedlot, beginning the feeding schedule carefully, providing sufficient, clean, fresh water, bone meal and trace mineral salt, and good shelter are far superior to any of the prophylactics."[14]

Mass use of tranquilizers has been found, on occasion, to be beneficial in certain aspects of beef cattle practice. It has been reported that tranquilizers can be econom-

ically feasible in reducing shrinkage in weaner calves.[2] Also, it has been found that tranquilizers have been helpful in reducing fence-walking and bawling in newly-weaned calves[2,5,7] and have facilitated feedlot adjustment and handling.[2]

It can be concluded from published information as well as from unpublished reports that most of the suggested mass uses of tranquilizers in beef cattle are of little or no benefit and on occasion may be detrimental.—A.F.

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Say It Better • • •

Watch out for words like *showed, exhibited,* and *demonstrated.* Grammatically and technically, these active verbs should not have inanimate subjects; seldom should they have non-human subjects in scientific writing.

To say that *the liver exhibited necrotic foci* is to ascribe to the liver a type of activity that even this marvelous organ cannot perform. Other unlikely events recently reported: inclusion bodies showed a basophilic tint; the spleen showed proliferation of cells; the radiograph demonstrated dysplasia; the colon showed degeneration; dogs exhibited inclusion bodies in their hepatic cells; the epidermis exhibited areas of edema; blood vessels showed cuffing; the dog exhibited vascular centers of organization; the animal showed an antibody titer of 1:256; the organs displayed dramatic lesions; the hemogram demonstrated leukopenia.

Actually, it's the investigator who does the showing or demonstrating. Why not say that the liver *was shown to have* necrotic foci, *there were* necrotic foci in the liver, the liver *was found to have* necrotic foci, or simply, that the liver *had* necrotic foci? All are better than having the liver show or exhibit.

If words like *showed* cause you trouble, try starting the sentence with *there was* or else make the lesion or the condition the subject of the sentence.

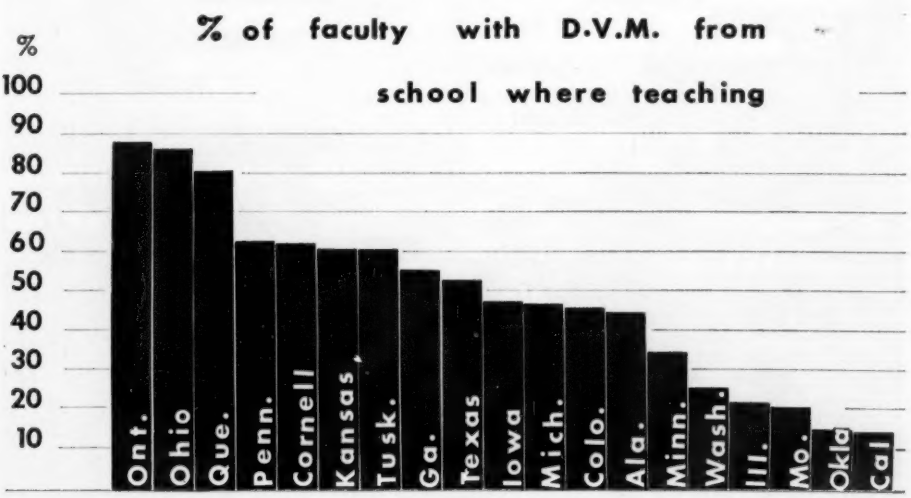
Per Cent of Faculty with D.V.M. Degree from Institution Where Presently Teaching

The number of veterinarians employed on faculties of veterinary colleges in the United States and Canada vary according to the size of the institution, the amount of research conducted, and the extent of service and extension programs. This number ranges from a low of 18 to a high of 66.

Some schools derive a high percentage of their faculty from the ranks of their own graduates, while at other schools nearly all faculty members have been trained elsewhere. The length of time a school has been established has some bearing on this factor; however, among the "older" schools the percentages are not uniform.

Per Cent of Faculty with D.V.M. Degree from Institution Where Presently Teaching

School	Per cent	School	Per cent
Ontario	89.1	Michigan	47.8
Ohio	86.6	Colorado	47.3
Quebec	80.7	Alabama	45.4
Pennsylvania	63.8	Minnesota	35.5
Cornell	63.6	Washington	26.0
Kansas	61.3	Illinois	22.5
Tuskegee	61.1	Missouri	20.8
Georgia	56.0	Oklahoma	16.2
Texas	53.6	California	15.5
Iowa	48.9		



AVMA Council on Education—1960

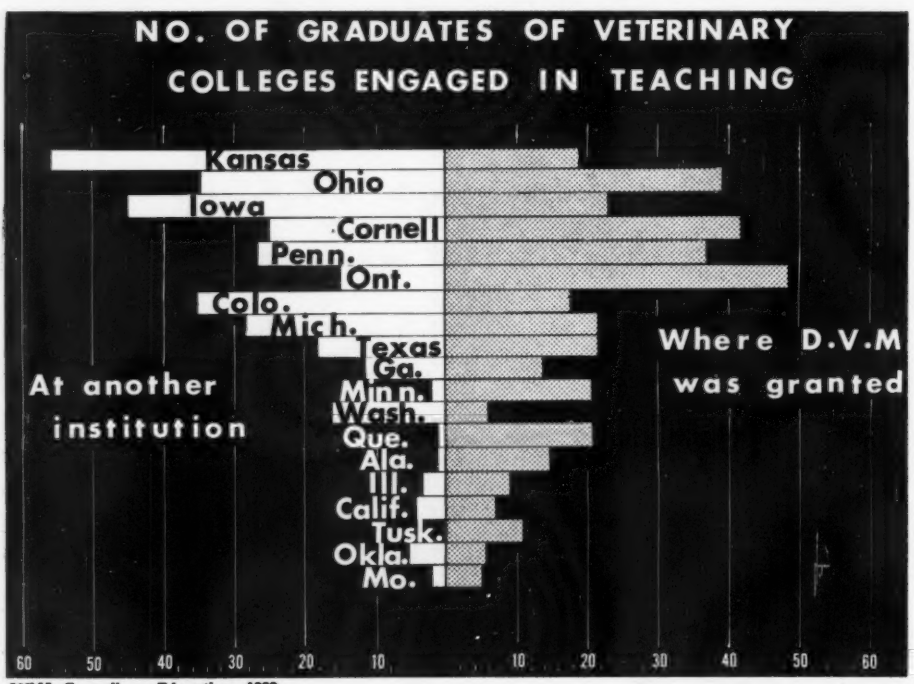
Number of Graduates of Veterinary Schools Engaged in Teaching

Of the 805 veterinarians on faculties at veterinary colleges in the United States and Canada, 715 (90%) are graduates of existing schools of these two countries; the remainder are graduates of schools that have been discontinued or are graduates of schools located in countries other than the United States and Canada.

About one-half of the graduates who engage in teaching do so at the institutions where they received their veterinary training.

Veterinarians Engaged in Teaching: School of Origin and Number Teaching at that School

Veterinary school	Number of graduates engaged in teaching at existing veterinary schools	Number teaching at institutions where degree was granted
Kansas	75	19
Ohio	74	39
Iowa	68	23
Cornell	67	42
Pennsylvania	64	37
Ontario	64	49
Colorado	53	18
Michigan	50	22
Texas	40	22
Georgia	25	14
Minnesota	23	21
Washington	22	6
Quebec	22	21
Alabama	16	15
Illinois	12	9
California	11	7
Tuskegee	11	11
Oklahoma	11	6
Missouri	7	5
Total	715	386



AVMA Council on Education—1960

from the Research Journal

Effect of Antimalarial Drugs on Fasciola Worms

The *in vitro* effect of some antimalarial drugs on fasciola worms of buffaloes, cattle, and sheep was studied, using a small glass jar bath. Quinacrine, amodiaquine, and pyrimethamine produced consistent stimulation of the motor activity of the worm. Quinine, however, produced stimulation of the parasite movement when added in small amounts, and stimulation preceded by inhibition when added to the bath in larger amounts. — [*A. Sharaf, M. H. Haiba, and I. M. Shihata: In Vitro Studies on the Effect of Some Antimalarial Drugs on Fasciola Worms in Buffaloes, Cattle, and Sheep. Am. J. Vet. Res., 21, (March, 1960): 308-310.*]

Hydroxycorticosteroids in Healthy Cattle

A total of 451 determinations of 17-hydroxycorticosteroids were made on the plasma of 46 cattle in various physiologic states. With the exception of estrous values of heifers and cows, no significant differences in plasma values were observed. No significant diurnal variations were ascertained in unbred, nonestrous heifers. — [*K. E. Shaw, S. Dutta, and R. E. Nichols: Quantities of 17-Hydroxycorticosteroids in the Plasma of Healthy Cattle During Various Physiologic States. Am. J. Vet. Res., 21, (Jan., 1960): 52-53.*]

Re-Exposure of Pregnant Sows to Leptospirosis

The immunity produced by active infection was sufficient to protect pregnant sows and their fetuses against challenge with a virulent strain of *Leptospira pomona*. Such animals should be satisfactory to retain for breeding purposes.

Active inflammatory processes appear to persist in the porcine kidney for 10 to 14 months after initial infection and 8 to 10 months after leptospires were demonstrated in the urine. — [*R. L. Morter, E. V. Morse, and R. F. Langham: Experimental Leptospirosis. VII. Re-Exposure of Pregnant Sows with Leptospira pomona. Am. J. Vet. Res., 21, (Jan., 1960): 95-98.*]

Carbon Tetrachloride Poisoning of Ewes

Ewes on a low selenium intake (0.5 p.p.m. of ration, dry weight basis) developed signs of poisoning and 1 died when 1 ml. of CCl_4 was administered orally. Characteristic lesions of CCl_4 poisoning, including hepatic centrolobular hemorrhagic necrosis, occurred in the ewe that died. Controls given 0.15 p.p.m. of selenium in the ration were unaffected by the same treatment.—[O. H. *Muth: Carbon Tetrachloride Poisoning of Ewes on a Low Selenium Ration. Am. J. Vet. Res., 21, (Jan., 1960): 86-87.*]

New Books

Artificial Insemination of Farm Animals

This third revision of this book, which had its first printing in 1945, has incorporated new material written by several international authorities.

New chapters include "Evaluation of Semen by Chemical Analysis" by Dr. T. Mann of Cambridge University; "Buffaloes" by Dr. P. Bhattacharya of India; "Frozen Semen" by Dr. H. A. Herman of the National Association of Artificial Breeders; and "Disease and Artificial Insemination" by Dr. David E. Bartlett and Dr. Lester L. Larson of the American Breeders Service.

Advances in new areas of artificial insemination in swine and poultry have been included. Dr. John Aamdal, Norway, has rewritten the chapter on swine.

Revisions have been made in the chapters on dogs, horses, and jackstock, the shipping of semen, and the role of hormones in reproduction.

The chapter, "Advantages and Limitations," has been omitted because artificial insemination is no longer a new field in need of evaluation.—[*The Artificial Insemination of Farm Animals. Edited by Enos J. Perry. 3rd rev. ed. 430 pages; illustrated. Rutgers University Press, 30 College Ave., New Brunswick, N.J. Price $6.50.*]

The Elements of Style

Excellent books have been written on grammar and style; however, the distinguishing characteristic of this volume is that the basic elements of good writing are given in fewer than 75 pages and in refreshingly simple, clear-cut prose.

Respect of rules, maintenance of simplicity, and elimination of the superfluous are stressed as keys to lucid composition. The authors believe that "vigorous writing is concise." In creating this brief, yet authoritative, book, they have followed their own tenet explicitly.—[*The Elements of Style. By William Strunk, Jr.; with revisions, an introduction, and a new chapter on writing by E. B. White. 71 pages. Macmillan Company, 60 5th Ave., New York, N. Y. 1959. Price $2.50.*]

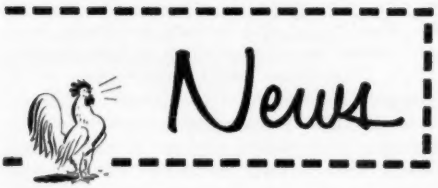
Diseases of Laboratory Primates

This is the first of 4 volumes entitled "Handbook of Primates." It reviews spontaneous diseases of laboratory primates, especially those diseases of parasitic or nutritional origin in monkeys. It describes conditions causing high mortality in monkeys: enteric diseases, respiratory infection, and tuberculosis. Attention is given, also, to numerous pathogenic agents accidentally transmissible to man, i.e., B-virus, and prevention of transmission.—[*Diseases of Laboratory Primates. By Theodore C. Ruch. 600 pages. W. B. Saunders Company, Philadelphia, Pa. 1959. Price about $7.50 (adapted from a review in Nature, 186, (May 14, 1960): 506.*]

Leaflet Available on Pesticides in Milk

An educational leaflet entitled "Keep Residues of Drugs and Pesticides Out of Milk" is now available on written request from the United States Department of Health, Education and Welfare, Food and Drug Administration, Washington 25, D. C.

It is intended to help dairy farmers, veterinarians, and others understand the importance of avoiding contamination of milk with drugs and pesticides and emphasizes the "Read the Label" theme as a preventive measure.



Applications for Fellowship Awards Reviewed

The AVMA Council on Research met in the central office of the AVMA with the following present: Officers T. Carl Jones, chairman; C. Roger Smith, secretary; and Council members C. A. Brandly, Robert Getty, W. A. Hagan, Rue Jensen, Hadleigh Marsh, R. D. Turk, and J. D. Wheat. Executive Secretary H. E. Kingman and Staff Consultant L. Meyer Jones were also present.

One of the principal activities of the Council on Research at this meeting was to review applications for renewal and new fellowship awards. The following six requests for renewal of fellowship awards were granted: Dr. Charles Reid for one year to study clinical radiology at the Cornell Medical School; Dr. E. W. Adams for one year to complete his studies in pathology at New York State Veterinary College; Dr. M. W. Glenn to complete studies in pathology at Colorado State University; Dr. C. L. L'Ecuyer to continue his studies in pathology at Iowa State University; Dr. G. Lussier for continued studies in pathology at Ontario Veterinary College of the University of Toronto; Dr. D. C. Secord for continued study in physiology at the same school. In addition, the Council is supporting Dr. A. Horowitz, currently studying anatomy at Iowa State University, by action at a previous meeting.

Regrettably, funds were available to grant fellowships to only four of 21 new applicants. The following four applicants were granted fellowships for one year of study at institutions of their request: Dr. R. L. Brinster, to study internal medicine at the University of Pennsylvania; Dr. R. C. Williams, to study pathology at New York State Veterinary College; Dr. Max Freeman, to study bacteriology at the University of Wisconsin; Dr. J. Godu, to study parasitology at the Ontario Veterinary College of the University of Toronto.

The Committee on Goals and Means suggested that a study leading to a report on the nature and extent of basic research and its relationship to the entire veterinary profession be initiated. The study should lay the basis for detailed consideration of the research program required to fulfill the profession's needs at the present time and in the future. The following projects are examples of those which should be considered:

1) Prepare for each species of domesticated animal various reports of investigation that have been accomplished and neglected.

2) Enumerate and categorize available research facilities throughout the country.

3) Set forth financial support and sources for research in veterinary medicine.

4) Estimate the amount of research on veterinary problems being conducted by nonveterinarians.

The Committee on Goals and Means believes that a nationwide collection of data on basic research policies, budgets, and expenditures should clarify the present status of research in veterinary medicine.

The report of this committee was accepted by the Council on Research.

The Committee on Liaison recommended acceptance of reports from representatives of the Council in attendance at various meetings. Dr. C. Roger Smith, who attended a meeting of the American Association for the Advancement of Science, submitted a request from that organization that the Council send a representative annually to its meetings and that the representative be elected for a term of three successive years. Dr. W. A. Hagan reported on a meeting sponsored by the U. S. Department of Agriculture for the purpose of eradicating hog cholera in the United States. The consensus of veterinarians and others in attendance at this meeting was that the time had come to "cease living with cholera" and to undertake a concerted federal-state program for eradication of the disease. Dr. C. K. White-

hair represented the Research Council at the Nutrition Council of the American Feed Manufacturers' Association. The Veterinary-Nutrition Relations Committee of the Nutrition Council adopted the following resolutions of interest to AVMA members:

1) The U. S. Livestock Sanitary Association is requested to appoint a representative to the Nutrition Council.

2) The Feed Additive section should be continued in the Veterinary Drug Encyclopedia and Therapeutic Index."

3) Veterinary diagnostic laboratories are extremely important to the livestock industry and the welfare of the people. These laboratories should be well staffed and coordinated with other state agencies.

The Committee on Liaison recommended acceptance of the report of the Committee on the Registry of Veterinary Pathology, which reported the activities of the Registry in providing special training in pathology for graduate veterinarians and in preserving, identifying, and making available special animal tissues of interest to pathologists.

The Committee on Liaison approved a proposal from the Agricultural Research Institute (NRC) submitted to various scientific and professional groups in the United States, that a conference be held on the general subject of effects of legislative and regulatory measures on research.

The Committee on the *American Journal of Veterinary Research* reported on many items concerning the publication, including the recommendation that summaries of articles in future issues of the *A.J.V.R.* be published in Interlingua in addition to English, on a trial basis for at least a year. Editor Price reported that approximately one-fourth of the copies of the *A.J.V.R.* now go to foreign countries.

New Veterinary Research Center at Wisconsin

The start of a $500,000 campaign to build and equip a veterinary science research center at the University of Wisconsin has been announced. The campaign is an official project of the University of Wisconsin Foundation.

Work of the Department of Veterinary Science is now performed in 3 separate buildings and other facilities scattered about the campus. This separation and crowding limits the training of graduate students and hampers research.

Actually, nearly $1½ million will be required to construct the building and provide equipment necessary for the University's veterinary science program. A grant of $694,000 has been recommended by the National Institutes of Health, U. S. Department of Health, Education and Welfare. This grant must be matched by nonfederal funds before it can be claimed.

About $200,000 already has been pledged toward matching the NIH grant. The remaining $500,000 is the goal of the campaign which is now being directed toward the chemical-pharmaceutical industry, the feed industry, the livestock and meat-packing industries, and veterinarians.—*Nat. Hog Farmer (July, 1960): 11.*

Fifth CDC Conference Scheduled for September

The fifth CDC biennial conference for public health veterinarians and teachers of preventive medicine will be held Sept. 12-17, 1960, at the new facilities of the U. S. Public Health Service's Communicable Disease Center in Atlanta, Ga.

Concurrent with the CDC meeting will be meetings of the Association of State Public Health Veterinarians and of the Teachers of Veterinary Preventive Medicine and Public Health.

Epidemiology and control of animal diseases pertinent to public health will be stressed. The major portion of the program will be devoted to discussions of the more important diseases of animals transmissible to man. Other sessions will encompass the newer developments in veterinary public health, such as mastitis, meat hygiene, comparative epidemiological investigations, and laboratory animal diseases.

In addition, there will be a review of the over-all Communicable Disease Center program.

The meetings are open, not only to public health veterinarians and teachers of veterinary preventive medicine, but also to research workers in comparative medicine and other interested persons.

Ralston Purina Fellowship Winners Announced, Dr. M. J. Freeman Receives Veterinary Honors

For 1960-1961, ten winners were selected to receive the Ralston Purina Research Fellowships by the Company's awards committee on March 28, in St. Louis, Mo.

In the field of veterinary medicine, Dr. Max James Freeman (AU '58) of Madison, Wis., was the winner. He will conduct his studies at the University of Wisconsin.

This is the twelfth year of the program. Each Award is in the amount of $1,800. The awards committee evaluates the applications on the basis of scholastic record, proposed research project, recommendations and other pertinent data relative to the ability of the applicant.

s/R. C. MORTON, *Manager, Educational Department.*

Among the States and Provinces

Alabama

ANNUAL CONVENTION OF ALABAMA V.M.A.—Mobile's Battle House Hotel was the site of the April 3-5, 1960, Alabama V.M.A. convention which was attended by 200 veterinarians and their wives.

Among the speakers on the scientific program were Drs. S. F. Scheidy, AVMA president; Iain M. Paton, Kansas City, Mo.; J. E. Greene, dean of the School of Veterinary Medicine, Auburn University; E. T. York, director of extension service of Auburn University; R. W. Storey, Muskegon, Mich.; W. G. Magrane, Mishawaka, Ind.; and Mark L. Morris, director of the Morris Foundation, Allen Park, Colo.

Dr. Ray Dunlap, Guntersville, became president, replacing retiring President Dr. Ross Cryar, Birmingham. Dr. W. P. Monroe, Anniston, is president-elect; Dr. Lawrence Cottle, Mobile, new member of the executive board; and Dr. M. K. Heath, Auburn, secretary-treasurer.

Delegates from the Alabama Medical Association, Alabama Dental Association, and Alabama Pharmaceutical Association attended some sessions of the convention at the invitation of the newly formed Interprofessional Council of Alabama.

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STATE ASSOCIATION HONORS STUDENT.—John Watts, Leesburg, Fla., senior student in veterinary medicine at Auburn University, was presented an award by the Alabama V.M.A. for saving a man's life at Auburn. Mr. Watts held closed the jugular vein of the injured man until he could be taken to a hospital.

California

MAYOR PROCLAIMS VETERINARY MEDICAL WEEK—San Francisco's Mayor George Christopher signed a proclamation designating Veterinary Medical Week in that city, saluting the 72nd annual convention of the California Veterinary Medical Association, June 27-29, 1960, at the Jack Tar Hotel.

The honor recognizes the veterinarian's achievements in maintaining the health and welfare of animals and his contributions to the health and welfare of man through progress in the prevention of diseases transmissible to man and through attention to animal products.

Mayor George Christopher (center), San Francisco, signs the proclamation of Veterinary Medical Week in the presence of Dr. R. L. Collinson (left), Modesto, program chairman, and Kenneth Humphreys, executive secretary, California V.M.A.

Georgia

ATHENS—CHAPTER OF PHI ZETA ESTABLISHED at UNIVERSITY OF GEORGIA.—On Nov. 13, 1959, the Xi Chapter of Phi Zeta, national honorary veterinary society, was organized on the campus of the University of Georgia. This is the fourteenth local chapter of this organization.

Charter members from the faculty, last year's graduating class, and present fourth year class were initiated. Present for the occasion were: Colonel W. E. Jennings, Office of the Surgeon General, Fifth Army Headquarters, Chicago; Dr. C. K. Mingle, Riverdale, Md.; Dr. H. L. Marsh, Princeton, Ill.; Dr. H. J. Stafseth, professor emeritus, Department of Microbiology and Public Health, Michigan State University, East Lansing, Mich.; and Dr. James R. Hay, AVMA director of professional relations.

Charter members are:

Olive Kendrick Britt ('59)
James Conrad Brown ('59)
James Robert Duncan ('59)
Joseph D. Edens ('52)
Charles C. Gue ('60)
Paul E. Hoffman (COR '53)
Thomas J. Jones ('33)
James Malcolm Kling ('59)
William A. Knapp ('51)
Sam S. Kreuz (TEX '42)
William Meredith Lee ('60)
Adrian M. Mills (COR '20)
Alvin F. Moreland ('60)
Paul L. Piercy (ISU '33)
Hugh Melvin Powell ('60)
Samuel C. Schmittle (OSU '47)
Frank D. Taylor ('60)
Ezekial F. Thomas, Jr. ('60)
Sara Jane Ulrich ('59)
Donald E. Weinman (KSU '46)
Clifford Westerfield (MSU '38)

The following officers were elected: Drs. Clifford Westerfield, president; Alvin F. Moreland, president-elect; Charles S. Gue, vice-president; Ezekial F. Thomas, Jr., secretary-treasurer.

S/C. WESTERFIELD, *Professor and Head Department of Anatomy and Histology.*

Illinois

DR. ERDHEIM RETURNS FROM EUROPE.—Dr. Morris Erdheim, Highland Park, Ill., who recently toured Europe, attended the annual meeting of the Israel Veterinary Medical Association at Tel Aviv, February 5-6, where he extended greetings from veterinarians in this country. The Israel Association has 158 members, including 3 women veterinarians.

Indiana

AMERICAN VETERINARY RADIOLOGY SOCIETY CONVENES.—The American Veterinary Radiology Society met Jan. 12, 1960, at the Severin Hotel, Indianapolis.

President William H. Rhoades, Philadelphia, Pa., called the convention to order and introduced the following program: Drs. Svend W. Nielsen, Columbus, Ohio—radiological aspects of bone tumors; John A. Campbell, Indianapolis, Ind.—thought control in roentgen diagnosis; W. H. Crago, Youngstown, Ohio—contrast mediums in radiological diagnosis; W. F. Riley, East Lansing, Mich.—radiological diagnosis of large animal lamenesses; and Myron Bernstein, Glencoe, Ill.—moderator of a film-reading session.

• • •

INDIANA V.M.A. MEETS IN 76TH CONVENTION.—Dr. L. A. Clark, president, Bedford, called the annual meeting of the Indiana V.M.A. to order for a three-day session Jan. 13, 1960, at the Severin Hotel, Indianapolis.

Speakers at the sessions on small animals included Drs. Robert Leighton, New York—surgical procedures for routine practice and open reduction for some hip conditions in the dog; Svend W. Nielsen, Columbus, Ohio—canine pancreatitis and disorders of canine rectal area; J. J. Fishler, Elkhart—radiological hazards; W. H. Crago, Youngstown, Ohio—inexpensive appliances; and George Burch, New Augusta—moderator of question-and-answer program. Presiding officers of these sessions were Drs. Frank Booth, Elkhart, and Robert Becker, Rensselaer.

Drs. Roy Bridge, Martinsville, and William Lamkin, Marion, presided over the large animal sessions. These programs included Drs. W. F. Riley, East Lansing, Mich.—equine practice; Charles B. Randall, Kinston, N. C.—swine practice and cattle practice; B. W. Kingrey, Ames, Iowa—cattle practice and Iowa State Clinic; Wilson Henderson, Lafayette—poultry industry; Robert Newlin, Columbus—moderator of large animal question-and-answer session; and John J. Updike, Elwood, and Richard Matteson, Brookeston—procine anemia. At the last afternoon session, Dr. Arthur Freeman, assistant editor of AVMA publications, extended greetings from the AVMA.

New York

DR. H. K. FULLER HONORED.—Dr. H. K. Fuller, Interlaken, was honored for 20 years of continuous service as technican for the Seneca Cooperative Cattle Breeders' Association, Inc., at the organization's twentieth anniversary celebration, Nov. 9, 1959. Dr.

Fuller is the first man to qualify for the NAAB Award.

Ohio

STATE V.M.A. TRAVELS "AROUND THE WORLD."—The 76th Annual Meeting of the Ohio State V.M.A. convened Jan. 31-Feb. 3, 1960, at the Deshler Hilton Hotel, Columbus.

"Around the World" was the theme of the social aspect of the program, with luncheons and other social activities with Spanish, German, and Oriental flavors.

Special and general sessions were run concurrently. The theme of the preventive medicine session was "Epidemiology in Veterinary Medicine." Dr. R. A. Masterson, Somerset, presided over the program which included Drs. Robert K. Anderson, St. Paul, Minn.—principles of epidemiology; H. Bradley Wells, Chapel Hill, N. C.—statistics; Charles Gale, Wooster—ornithosis; Earl J. Catcott, Cincinnati—respiratory diseases; and F. B. Clack, Pittsburgh, Pa.—leptospirosis. The general session, Dr. Karl S. Grady, Cincinnati, presiding, included Drs. C. L. Blakely, Boston, Mass.—surgical treatment of chronic ear infections; Walter A. Venzke, Columbus—hormones; Chauncey Leake, Columbus—cooperation among health professions; Robert P. Knowles, Miami, Fla.—cosmetic approach to surgery; Ralph C. Belding, East Lansing, Mich.—poultry disease problems; and Richard S. Witter, Columbus—equine diseases.

The small animal session, the theme of which was "Canine Thoracic Surgery," was presided over by Dr. V. G. Crago, Youngstown. Participants were Drs. C. R. Smith, Columbus—respiratory physiology; Robert P. Knowles—anesthesia and artificial respiration; Robert Hamlin, Columbus—cardiac emergencies and cardiac anatomy and diagnostic procedures; Svend W. Nielsen, Columbus—pathology; and H. William Clatworthy, Columbus—experimental esophageal surgery.

Dr. Nelson King, Wooster, presided over the concurrent general session and presented Drs. Harold Amstutz, Columbus—displacement of the abomasum (film); Charles Gale, Wooster—epidemiology of shipping fever; H. G. Headly, Camden—respiratory problems in swine; R. D. Radeleff, Kerrville, Texas—toxicology of insecticides; and Earl M. Baldwin, Omaha, Neb.—bovine infectious ketosis.

The final special session had "Nutrition in Modern Large Animal Practice" as its theme and was presided over by Dr. James Bratton, Athens. The four speakers included Drs. Charles E. Jordan, Indianapolis, Ind.—swine feeding; Robert Marshak, Wallingford, Pa.—nutritional aspects of metabolic diseases of cattle; Earle W. Klosterman, Wooster—beef cattle and sheep nutrition; and Walter A. Venzke, Columbus—hormones and nutritional diseases.

The final general session's presiding officer was Dr. James L. Stansbury, Marietta. This session featured Drs. W. E. Wendt, Cleveland—care of unusual pets; F. B. Clack, Pittsburgh, Pa.—epidemiology of dog bites; Paul Schnurrenberger, Columbus—rabies control in Ohio; Edward F. Donovan, Columbus—new therapeutic approach to fungus diseases; and C. L. Blakely, Boston, Mass.—fenestrations for cervical disk diseases in the dog.

Texas

DR. R. M. ZIRKLE GETS DEGREE IN LAW.—Dr. R. M. Zirkle (OSU '40) of San Angelo, Texas, graduated in May, 1960, from the law school of St. Mary's University of San Antonio, magna cum laude. Formerly a practicing veterinarian, Dr. Zirkle was assigned to the San Antonio Union Stockyards by the Agricultural Research Service several years ago. He studied for his law degree at night.

Prior to the ARS employment, Dr. Zirkle had been in the ranching and stock farming business, had conducted an equine practice, and had served on three occasions in the armed forces.

Vermont

DR. JOHN CANTY RETIRES.—Dr. John Canty, Montpelier, was honored at a testimonial dinner in recognition of his 41 years of service to the Vermont Department of Agriculture. Dr. Canty was the first full-time veterinarian hired by the state department. He was active in the tuberculin testing program and saw Vermont become a modified tuberculosis-free state in 1936. He was appointed state veterinarian and chief of the livestock division in 1947. After his appointment, Vermont became a modified certified brucellosis-free state. He has served as president of the Vermont V.M.A. and is a member of the AVMA.

Graduating Class, 1960, Ontario Veterinary College, University of Toronto

Top row (right to left)—P. Kushnerenko, E. A. Janzen, L. J. Banbury, W. Combe, G. J. Losos, B. Mufford, G. V. M. Mowbray, J. S. Browne, R. Skoropad.
Second row—D. F. Dineen, H. Sutmoller, V. West, C. Van Breemen, Dr. C. K. Roe, C. K. Yeo, R. A. Watt, D. MacKay, J. H. Lumsden.
Third row—W. R. Lawless, K. F. Choy, A. Bildfell, H. Geissinger.
Fourth row—G. Jones, D. Moore, B. Riehl, P. W. Wybenga, D. D. Stimpson, R. D. Axelson, R. J. Black, L. K. Anderson, J. A. Sankey, C. C. Gay.
Fifth row—V. M. Mohabir, D. C. Lund, A. J. Mowbray, A. Kahn, T. Poelma, R. Darling, P. Ide, J. E. Alexander, E. Greselin, H. Bacon.

University of Toronto.—At the 1960 commencement exercises of the Ontario Veterinary College, University of Toronto, the following 39 candidates were presented for the D.V.M. degree:

John E. Alexander
Lennart K. Anderson
Richard D. Axelson
Harry A. Bacon
Lloyd J. Banbury
Albert Bildfell
Robert J. Black
James S. Browne
Kwai F. Choy
William W. Combe
Ronald A. Darling
David F. Dineen
Clive C. Gay
Hans D. Geissinger
Erminio Greselin
Peter R. Ide
Elmer A. Janzen
Glen A. Jones
Awal Khan
Peter Kushnerenko
William R. Lawless
George J. Losos
John H. Lumsden
Darwin C. Lund
Donald W. Moore
Alison J. Mowbray
Glen V. M. Mowbray
Barry K. Mufford
Robert D. MacKay
Theodoris A. Poelma
Benson A. Riehl
John A. Sankey
Don D. Stimpson
Hugo Sutmoller
Cornelis Van Breemen
Ralph A. Watt
Vernon S. West
Pieter W. Wybenga
Chwee K. Yeo

Graduating Class, 1960, School of Veterinary Medicine, A. & M. College of Texas

Top row (left to right)—R. E. Ables. Ables, A. R. Allbritton, J. L. Barkley, J. R. Barlow, L. T. Baron, F. L. Becker, T. A. Beckett, J. R. Berryman, J. O. Brumlow, B. J. Cargill.

Second row—A. B. Childers, Jr., D. R. Clark, A. I. Davidson, N. K. Downard, W. R. Dudley, Jr., C. A. Eckhardt, J. H. Farris, Jr., J. W. Foster.

Third row—L. M. Fuson, S. E. Glass, J. D. Gleason, D. E. Greenberg, N. B. Guilloud, R. C. Hall, S. M. Halley, D. F. Houston, J. L. Howard , J. F. Hubbs, Jr.

Fourth row—C. L. Jay, J. E. Jordan, W. C. King, T. L. McLaughlin, Louis Martin, C. D. Minnis, B. M. Muirhead, R. M. Nance, A. D. Nelson, S. G. Nolte, Jr.

Fifth row—T. M. Perkins, J. B. Phillips, Ed Pigott, Jr., H. D. Putnam, S. H. Ridgway, J. E. Sandusky, O. E. Schroeder, L. B. Sells, W. G. Shelton, Alton F. Smith.

Sixth row—David L. Smith, Perry Smith, A. L. Speck, J. P. M. Syler, R. B. Terry, R. J. Thomes, J. B. Tucker, Jr., J. T. Vance, III, R. E. Whitmire, Bob Wilson.

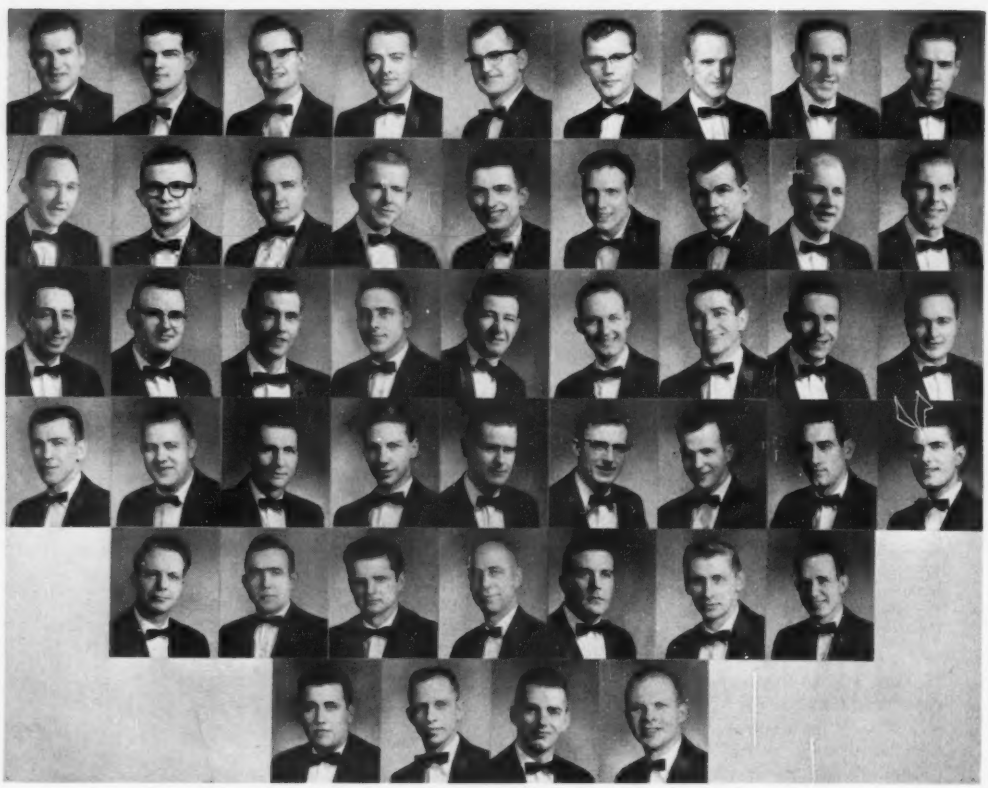
A. & M. College of Texas.—At the 1960 commencement exercises of the School of Veterinary Medicine, A. & M. College of Texas, the following 57 candidates were presented for the D.V.M. degree:

R. E. Ables
A. R. Allbritton
J. L. Barkley
J. R. Barlow
L. T. Baron
F. L. Becker
T. A. Beckett
J. R. Berryman
J. O. Brumlow
B. J. Cargill
D. R. Clark
A. B. Childers
A. I. Davidson
N. K. Downard
W. R. Dudley
A. C. Eckhardt
J. H. Farris
J. W. Foster
L. M. Fuson
S. E. Glass
J. D. Gleason
D. E. Greenburg
N. B. Guilloud
R. C. Hall
S. M. Halley
D. F. Houston
J. L. Howard
J. F. Hubbs
C. L. Jay
J. E. Jorden
W. C. King
T. L. McLaughlin
Louis Martin
C. D. Minnis
B. M. Muirhead
R. M. Nance
A. D. Nelson
J. G. Nolte
T. M. Perkins
J. B. Phillips
E. L. Pigott
H. D. Putnam
S. H. Ridgway
J. E. Sandusky
O. E. Schroeder
L. B. Sells
W. G. Sheldon
A. F. Smith
D. L. Smith
P. G. Smith
A. L. Speck
J. P. Syler
R. B. Terry
R. L. Thomes
J. B. Tucker
J. T. Vance
R. E. Whitmire
R. L. Wilson

Washington State University.—At the 1960 commencement exercises of the college of Veterinary Medicine, Washington State University, the following 47 candidates were presented for the D.V.M. degree:

William G. Albro
John E. Alman
Jack N. Armstrong
Alfred W. Bailey
Lloyd R. Beal
Bruce E. Belshaw
Joe D. Bergevin
James M. Berry
Rodger L. Blue
William F. Brown
Gary M. Bryan
James D. Burns
Douglas Y. Campbell
Charles C. Capen
Alvin T. Carver
Robert W. Chase
Arthur E. Fulkerson
Richard A. Fussell
Roger M. Gardner
Norman L. Harding
John W. Harrer
Richard E. Hazen, Jr.
William H. Henderson
John D. Hill
Robert H. Hogan
Herman C. Hopf, Jr.
Frederic W. Kullenberg
Jerry A. LaFollette
Robert E. Leid
Ernest T. Littledike
Roger O. McClellan
Ronald G. Middaugh
William H. Morton, Jr.
Patrick D. O'Callaghan
Robert D. Painter
Richard L. Perkins
William M. Porter
Glenn B. Rice
Ira D. Rudd
Elmer S. Sniff
Arthur E. Staudt
Thomas W. Weiger
George M. Wells
Robert M. Weston
Franklin K. Whitener
Robert W. Yates
Mark M. Young

Graduating Class, 1960, College of Veterinary Medicine, Washington State University



Top row, left to right—W. G. Albro, J. E. Alman, A. W. Bailey, J. N. Armstrong, L. R. Beal, R. L. Blue, B. E. Bleshaw, J. D. Bergevin, J. M. Berry.

Second row—W. F. Brown, G. M. Bryan, J. D. Burns, D. Y. Campbell, C. C. Capen, A. T. Carver, R. W. Chase, A. E. Fulkerson, R. A. Fussell.

Third row—R. M. Gardner, N. L. Harding, J. W. Harrer, R. E. Hazen, Jr., W. H. Henderson, J. D. Hill, R. H. Hogan, H. C. Hopf, Jr., F. W. Kullenburg.

Fourth row—J. A. LaFollette, R. E. Leid, E. T. Littledike, R. G. Middaugh, W. H. Morton, Jr., R. O. McClellan, P. D. O'Callaghan, R. D. Painter, R. L. Perkins.

Fifth row—W. M. Porter, G. B. Rice, I. D. Rudd, E. S. Sniff, A. E. Staudt, T. W. Weiger, G. M. Wells.

Sixth row—R. M. Weston, F. K. Whitener, R. W. Yates, M. M. Young.

Deaths

Star indicates member of AVMA

★**William A. Browne** (KSU '28), 67, Merced, Calif., died after a heart attack, Sept. 30, 1959.

Dr. Browne was a past-president of the Northern San Joaquin Valley V.M.A., and a member of the California V.M.A. and the AVMA.

★**William A. Drummond** (MSC '42), 41, was killed April 14, 1960, when his car was struck and overturned at Torrance airport near Los Angeles.

Dr. Drummond moved to the West Coast seven years ago from Detroit, Mich. He owned and operated the Hawthorne Small Animal Hospital in Lawndale, Calif.

★**Dave C. Dunbar, Jr.** (GA '58), 27, died in Bethesda, Md., after a long illness.

Dr. Dunbar, a first lieutenant in the U.S. Air Force, was stationed at Andrews Air Force Base, Md. Prior to this assignment in March 1959, he had practiced with Dr. Felix Smith in Perry, Ga.

Kenneth C. Farley (KSU '22), Medford, Ore., died Feb. 26, 1960, from a heart attack.

Following graduation, Dr. Farley practiced at Clarks, Neb., where he continued as a general practitioner until 1937. At that time, he entered the former BAI and was assigned to Oregon serving successively at John Day, Mount Vernon, and since 1940, at Medford. He was under consideration for promotion at the time of his death.

★**John R. Fuller** (KSU '12), 69, Walla Walla, Wash., died March 19, 1959.

Dr. Fuller was made a life member of the AVMA in 1956. He is survived by a son, Dr. Jay R. Fuller (WSC '50), also a veterinarian in Walla Walla.

Oscar Hartnagel (ONT '00; McK '01), 88, Van Nuys, Calif., died in Jan., 1960.

Frank E. Haworth (IND '07), 81, Arkansas City, Kan., died Dec. 12, 1959.

A retired government meat inspector, Dr. Haworth entered the meat inspection service in Chicago in 1907 and later worked in Indianapolis. He served two years in Oklahoma City and five years in Fort Worth before moving to Arkansas City in 1920. Dr. Haworth retired from there in January, 1940.

★**Thomas E. Head, Jr.** (API '43), 40, was killed in an auto accident March 15, 1960, at Denham Springs, La.

Dr. Head was employed by the ARS Animal Disease Eradication Branch in Louisiana and lived in Hammond.

He was a member of the AVMA, Alpha Psi, Alabama V.M.A., Louisiana V.M.A., Alabama Farm Bureau, and Louisiana Farm Bureau.

Bert E. Helms (IND '11), 78, died in Noblesville, Ind., Jan. 5, 1960, after a long illness.

Dr. Helms had retired from practice in Fortville; he was a member of the Indiana State V.M.A.

Ivan G. Howe (COR '14), 67, Belmont, N.Y., died in Buffalo, Jan. 20, 1960.

Dr. Howe was retired; he had served as director of the New York State Bureau of Animal Husbandry.

A. M. Kennelly (CVC '15), 69, Yates City, Ill., died following an illness of several weeks' duration on Feb. 27, 1960.

Except for the 19 years when Dr. Kennelly was supervisor of the state tuberculosis and brucellosis divisions, he had conducted his practice in Yates City. A brother, Dr. J. A. Kennelly (CVC '14), resides in Knoxville,

★**Chester M. Merrill** (USC '13), 71, died April 14, 1960, in South Paris, Maine.

Dr. Merrill practiced veterinary medicine in South Paris for 47 years. His son, Dr. Stanford D. Merrill, Augusta, Maine, is also a veterinarian.

★**John F. Planz** (OSU '04), 84, died May 18, 1960, in Portage Lakes, Ohio.

Dr. Planz was formerly president of the Ohio State V.M.A. and had retired from practice about 10 years ago.

★**George O. Smith** (ONT '00) 83, died from a coronary occlusion, in February, 1958, in Ligonier, Ind.

Dr. Smith was a life member of the Michiana V.M.A. and the AVMA (1954).

John M. Tritschler (UP '18), died March 8, 1960, in Cincinnati, Ohio.

Dr. Tritschler's father, Dr. Morris W. Tritschler, and his grandfather were also veterinarians.

Women's Auxiliary

California

CALIFORNIA—AUXILIARY MEETS IN SAN FRANCISCO.—Highlights of the June 26-27 meeting of the Women's Auxiliary to the California V. M. A. were initiated with the executive committee meeting at the Jack Tar Hotel in San Francisco. The local presidents met with the state officers.

A bus trip called the "Bay Cruise," lunch at Fisherman's Wharf, a cable car ride, a cocktail party, and the president's banquet filled the second day's activities.

Registration, the business meeting, and a joint meeting with the V. M. A. on business management completed the third day's agenda.

The final day, a luncheon featuring Count Marco, reputed as a "Continental sophisticate, man of the world, columnist, and authority of women, love, beauty, marriage, and husbands," an ice ballet, installation of officers, and the drawing of door prizes culminated the meeting.

Mrs. W. L. Kanawyer was chairman of the committee on local arrangements and was assisted by Mrs. Ernest A. Siegel, registration; Mrs. Richard L. Stowe, door prizes; Mrs. Norman Freid, decorations; Mrs. Roger A. Burr, secretary; Mrs. Howard F. Carroll, teenage program; Mrs. Milton C. Levy, Bay cruise; and Mrs. Edward C. Bland, decorations. They are all residents of San Francisco.

Mrs. Russell P. Cope, Berkeley, president-elect, worked with the men's association and the local committee as program coordinator.

Illinois

The Women's Auxiliary to the Central Illinois V.M.A. met April 18, 1960. Following dinner with their husbands, the 23 members present adjourned for their business meeting.

The meeting was called to order by the president, Mrs. Morland. The minutes for the last meeting were read and approved and the treasurer's report was read and filed for audit.

Mrs. Miller, Mrs. Smith, and Mrs. Starkey were members appointed by the executive board as a committee to revise the constitution. Copies of the suggested revision were given to each member. Mrs. Miller explained the changes at the Dec. 10, 1959, meeting. At this April meeting it was moved and seconded that the constitution and by-laws be accepted as revised. The motion carried.

The Auxiliary contributed $10.00 to the Student Loan Fund and $10.00 to the Research Fund. The president announced the appointment of Mrs. Darrell Jessen as membership helper.

Mrs. Morland announced her resignation as president and as delegate to the 1960 National Convention. Mrs. Folkerts, vice-president, will assume the office of president. Mrs. Jessen, alternate delegate, will represent the Auxiliary in Denver.

s/MRS. H. F. BENNETT, *Secretary.*

Wyoming

WYOMING—AUXILIARY HOLDS ANNUAL MEETING.—The student Union Building of the University of Wyoming, Laramie, was the site of the annual meeting of the Women's Auxiliary to the Wyoming V.M.A. on June 20, 1960.

Officers elected at the meeting are Mrs. Bert Reinow, Pinedale, president; Mrs. Robert Baldwin, Gillette, first vice-president; Mrs. Brinton Swift, Buffalo, second vice-president; Mrs. George Glover, Torrington, treasurer; Mrs. W. R. Lee, Powell, secretary; and Mrs. Robert Fuechsel, Riverton, historian.

s/MRS. W. R. LEE, *Secretary*

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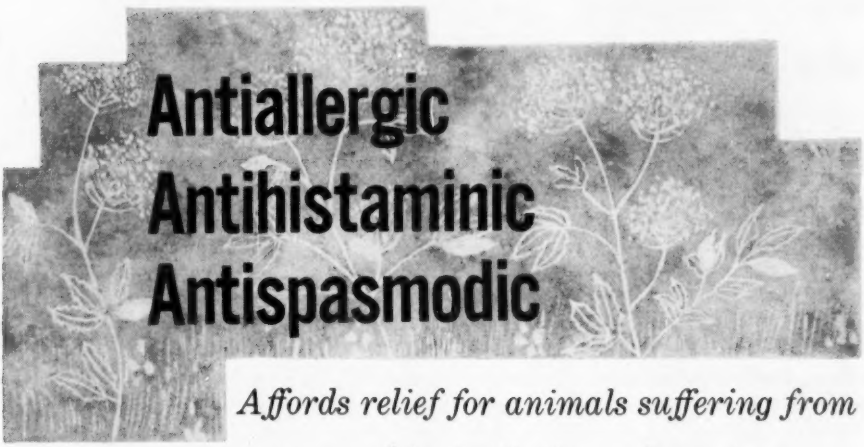

Antiallergic
Antihistaminic
Antispasmodic

Affords relief for animals suffering from common skin irritations, dermatoses and a variety of allergic conditions.

Benadryl Hydrochloride is available in a variety of convenient forms including: Kapseals,® 50 mg. each; Capsules, 25 mg. each; Emplets,® 50 mg. each, for delayed action; Elixir, 10 mg. per 4 cc.; Steri-Vials,® 10 mg. per cc. for parenteral use; and a Cream (2% Benadryl Hydrochloride).

PARKE-DAVIS

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DETROIT 32, MICHIGAN

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MONTREAL 9, QUEBEC

in small animals

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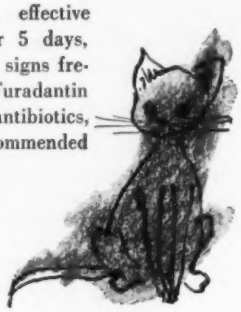
breaks the resistance barrier to clinical recovery

URINARY TRACT INFECTION: FURADANTIN therapy achieved clinical recovery in over 90% of the cases of acute or chronic urogenital disease in dogs and cats.[2] Pronounced symptomatic improvement occurred by the 4th day and complete recovery within 7-14 days.[3] Coles states, "One of the advantages of nitrofurantoin [FURADANTIN] in the treatment of canine nephritis is the fact that most microorganisms do not develop resistance to the drug, even after long periods of exposure to it."[4]

INDICATIONS: nephritis, cystitis, pyelonephritis; pre- and postoperative care of the urethra and bladder; prophylaxis in catheterized patients; and as an adjunct to surgical drainage in canine prostatic abscess.

CANINE TRACHEOBRONCHITIS: FURADANTIN constitutes modern, effective treatment for dogs with "kennel cough". When given orally for 5 days, FURADANTIN stopped coughing in 95% of 75 cases. In some animals, signs frequently subsided in 48 hours.[5] Mosier concludes, "In our experience, Furadantin has been considerably more effective than the various bacterins, antibiotics, vaccines, iodides, and chemotherapeutic agents which have been recommended for the treatment of tracheobronchitis in the past."[5]



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FURADANTIN Ora-Bols® Veterinary, provides 50 mg. FURADANTIN in an excipient containing dextrose. Bottle of 100, scored, 50 mg. Ora-Bols.

FURADANTIN Tablets Veterinary, bottles of 100, scored, 10 mg., and 100 mg. tablets.

FURADANTIN Oral Suspension Veterinary; provides 5 mg. FURADANTIN in each cc. Bottles of 60 cc.

Available through your professional veterinary distributor

Nitrofurans—a *unique* class of antimicrobials—neither antibiotics nor sulfonamides

Ora-Bols is the Eaton tradename for small, bolus-shaped tablets.

Eaton

EATON LABORATORIES, NORWICH, NEW YORK

REFERENCES: 1. Michaelson, S. M., and Covert, M.: J. Am. Vet. M. Ass. 134:334 (Apr.) 1959. 2. Mosier, J. E., and Coles, E. H.: Vet. Med. 53:649 (Dec.) 1958. 3. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 4. Coles, E. H., and Mosier, J. E.: Am. J. Vet. Res. 20:1020 (Nov.) 1959. 5. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

WHAT IS YOUR Diagnosis?

Make your diagnosis from the picture below—then turn the page

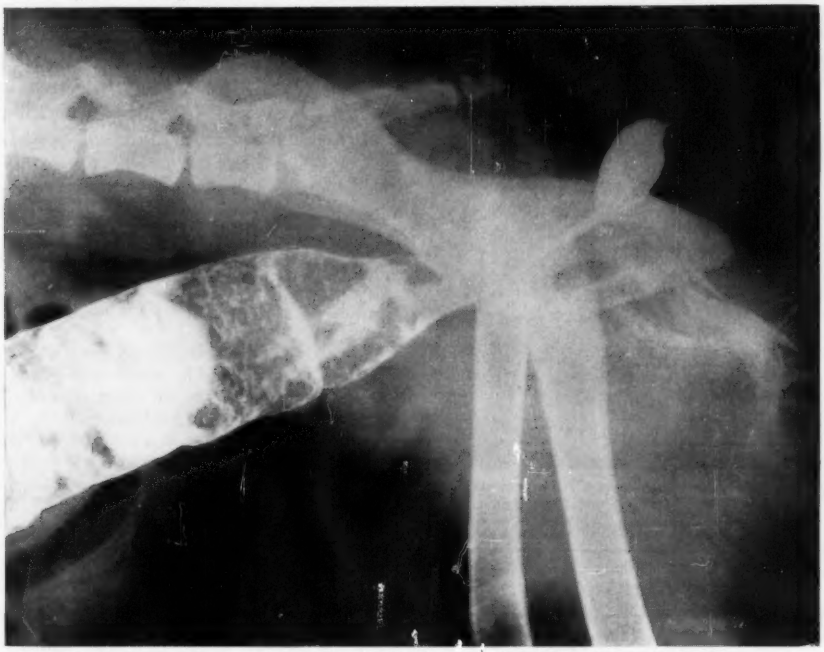

Fig. 1—Lateral recumbent radiograph of the Springer Spaniel, taken after barium enema.

History.—A male Springer Spaniel, 8 years old, had rectal tenesmus for 3 days. Since this dog habitually scavenged and ate bones, the owner assumed that the trouble was an impaction and brought the dog to the hospital for treatment. Upon examination, the rectum was found to be empty but was obstructed dorsally by a large, tender, firm mass situated ventral to the sacral and coccygeal vertebrae. A 16-gauge hypodermic needle was inserted through the skin into the mass and a cubic centimeter of hemorrhagic serum containing fat droplets was aspirated. The dog was given a barium enema and a lateral recumbent radiograph was taken (fig. 1).

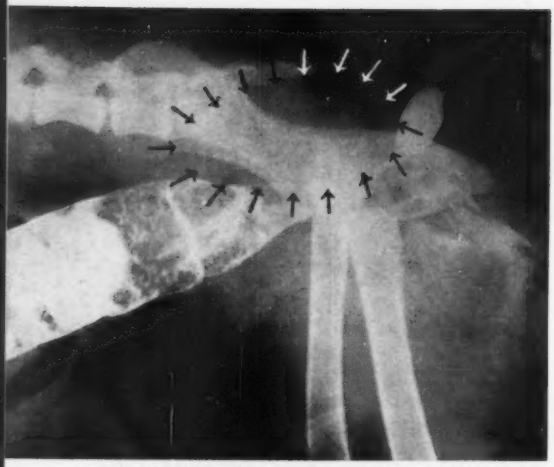

Fig. 2—Lateral recumbent radiograph of the Spaniel, showing the outline of the tumor obstructing the rectum (arrows).

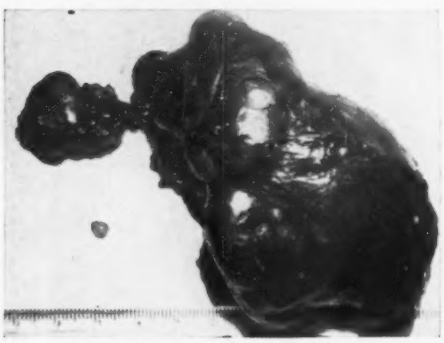

Fig. 3—Photograph of lipoma after removal.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Rectal obstruction due to a pelvic mass dorsal to the rectum and ventral to the sacral and coccygeal vertebrae (arrows, fig. 2).

Comment.—A fatty, hemorrhagic mass (fig. 3) measuring 9 by 6 cm. was removed without difficulty through an incision in the skin in the left perineal area. It did not seem to be connected to the prostate or the urinary bladder. Histologically, this lipoma was composed of blood, many leukocytes, and fat, some of which was necrotic. Undoubtedly the result of trauma, it provided evidence of inflamation, hemorrhage, and degeneration. Lipomas are not unusual in the subcutaneous or visceral areas where fat is normally found; when they become large enough to be subjected to trauma, the result is inflammation, edema, hemorrhage, or degeneration.

This case report was submitted by S. R. Roberts, D.V.M., Richmond, Calif., and was prepared with the assistance of Wayne H. Riser, D.V.M., M.S., Kensington, Md.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

Federal-State Program to Eradicate Cattle Fever Ticks Underway in Florida

Cattle fever ticks, found recently in Florida for the first time since 1957, are the target of a federal-state eradication program now under way.

State quarantine was placed on a triangular area on the eastern coast of Florida, involving parts of Martin and Palm Beach counties, and on two premises in Hillsborough County, following the discovery of tick infestations. All three counties were placed under federal quarantine on July 1.

State and federal inspectors are checking all cattle and horses in the quarantined areas. They also are tracing the movements of animals shipped from these areas to points in Florida and other states during the past several months, in efforts to prevent spread of the ticks.

The last outbreak of cattle fever ticks in Florida occurred in 1957, when 15 infested herds were found in Okeechobee, Broward, Dade, Highlands, and Palm Beach counties. Quarantines in these counties were lifted in September, 1958, after the last known infestations had been eradicated.

Until the current outbreak in Florida, the only tick-infested area in the United States has been a narrow buffer zone in parts of eight counties in Texas along the lower Rio Grande River. This buffer zone, adjoining tick-infested areas in Mexico, is also under quarantine.

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REFERENCES: Teigland, M. B.: Proceedings of the 4th Annual Meeting, Amer. Assn. of Equine Pract., Chicago, Illinois, 1959. Witter, R. S.: Paper read at the Third Regional Conference on the Nitrofurans in Veterinary Medicine, Atlanta, Georgia, January 14, 1960.

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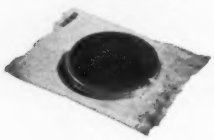

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History of the AVMA

1892

The 1892 meeting was held in Boston. Once a stronghold of Association activities, this was to be the last meeting there for more than 50 years.

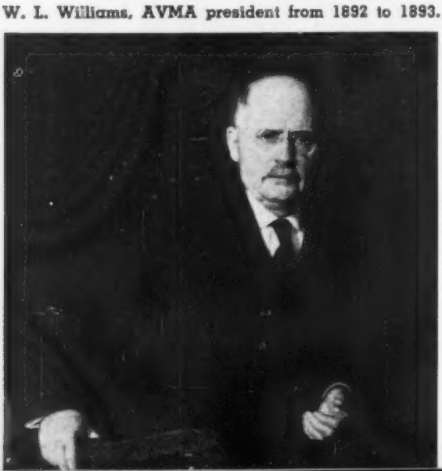
A major step was taken in unanimously adopting an amendment requiring that any future applicant for membership "shall be a graduate of a regularly organized and recognized veterinary school, which shall have a curriculum of at least three years, of six months each, specially devoted to the study of veterinary science, and whose corps of instructors shall contain at least four veterinarians." Despite Liautard's earlier opposition to the 3-year curriculum, he comments on the Association's stand: ". . . . today it has not only become the accepted representative body of the veterinary profession of America, but has acquired such a weight of authority as to enable it to initiate and establish a movement towards which for years many veterinarians have vainly (because singly) worked, to wit: the establishment upon a permanent foundation of an advanced and uniform standard of education for veterinarians in the United States."

The admission of 101 new members marked the largest accession in the history of the Association—more, in fact, than comprised the total active membership less than a decade earlier. W. L. Williams was elected president, and A. W. Clement, vice president, W. H. Hoskins was re-elected secretary, and J. L. Robertson, treasurer.

Papers were presented by Olaf Schwartzkopf and W. L. Williams on food inspection, and by Williams on veterinary science in agricultural colleges. Speaking on the work of the BAI, D. E. Salmon reminds his listeners: ". . . . it is one of the objects for which this Association exists, to promote and encourage scientific research . . . . and that any criticisms will be fair and truthful . . . to bring out points which may have been obscure, or to call attention to conclusions which may be untenable. . . . Our great need is that we should have more original investigations."

Salmon was rankling over a report issued by the Committee on Intelligence and Education, which severely criticized the work of the Bureau as not having a "creditable appearance to other civilized nations," and commended Billings' work. Salmon characterized the report as ". . . . misrepresentation . . . calculated to bring reproach upon our Department of Agriculture, our scientists, our institutions, and our country . . . . to retard the progress of science." A majority of the profession sided with Salmon by this time. Billings had been expelled from the Association two years earlier.

W. L. Williams, AVMA president from 1892 to 1893.



✦ ✦ ✦

WALTER LONG WILLIAMS, V.S., was born in 1856 near Argenta, Illinois. After teaching in a rural school 2 years, he entered the Illinois Industrial University (now Univ. of Ill.) in 1875. Transferring to the Montreal Veterinary College, his tutelage under William Osler was an inspiration for his later painstaking studies in the problems of reproductive pathology. In the 1880's he recognized dourine in horses in Illinois and was largely responsible for much of our knowledge of this disease. In 1896 he became Professor of Veterinary Surgery, Obstetrics, Zootechnics and Jurisprudence at Cornell University, where he was responsible for many innovations in veterinary teaching until his retirement in 1921.

A prolific writer, he contributed many articles to the veterinary journals both here and abroad, and was the author of two classic texts, *Veterinary Obstetrics,* and *Diseases of the Genital Organs of Domestic Animals,* both familiar to most veterinarians. Active in Association affairs, Dr. Williams served as president of the Illinois VMA (1889), USVMA (1892-93) and the N.Y. Veterinary Medical Society (1906, 1907). After retiring from teaching he remained active in professional affairs and was a famliar figure on the Cornell campus until shortly before his death October 23, 1945, at the age of 89.

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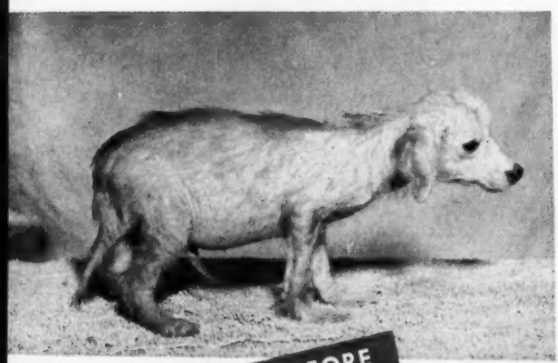

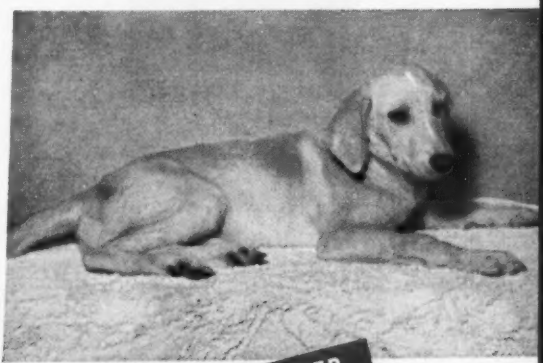

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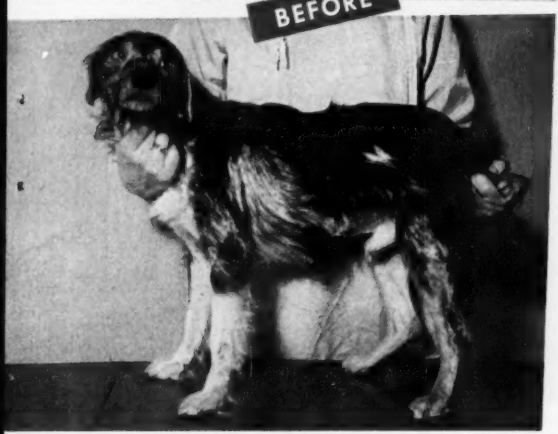

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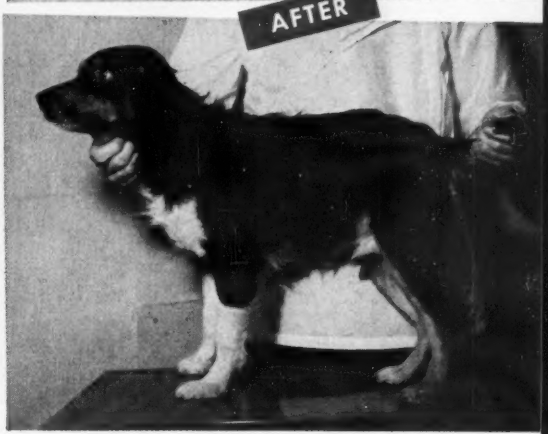

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1. "Clinical Evaluation of a Drug for Dermatoses of Dogs and Cats," Wenger, J. B., Vet. Med., 55:55-58, Mar. 1960.

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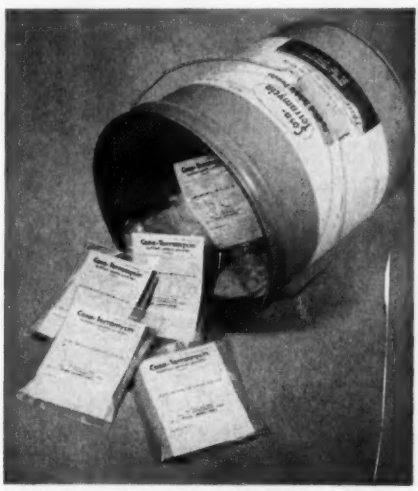

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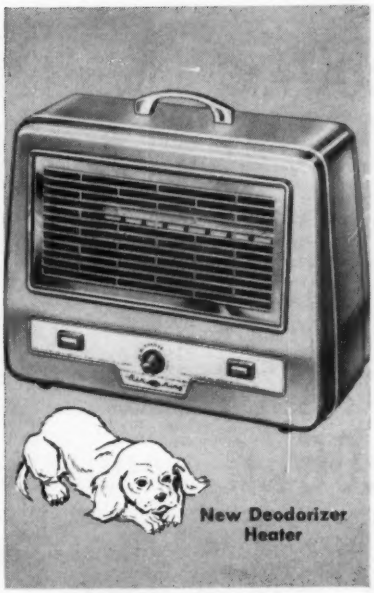

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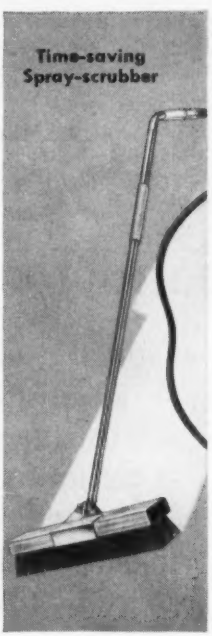

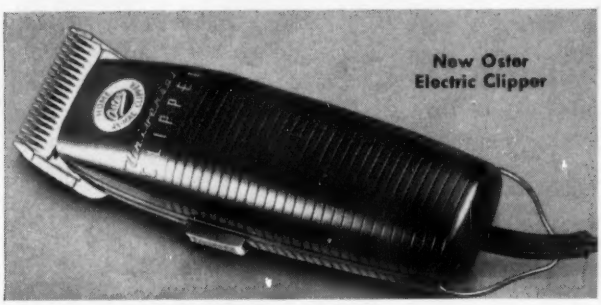

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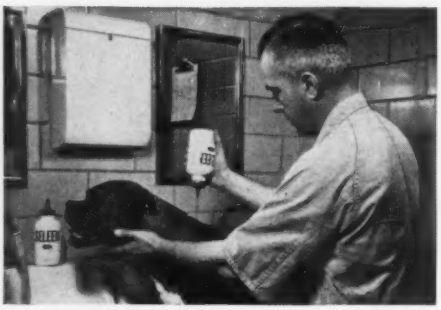

2:02 P.M. Easy Doc, that spot's a little tender.

2:06 P.M. Ahhh. Just a little higher and to the right.

2:12 P.M. I like it. Seleen. Be with you in a minute.

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2. What effect did the tranquilizer triflupromazine have on calves at weaning and during transit? Page 240.

3. What is Christmas disease and of what importance is it in dogs? Page 250.

4. What are the optimum preanesthetic dosages in cats of the tranquilizers promazine and promethazine when given in conjunction with pentobarbital sodium anesthesia? Page 253.

5. What is toxic fat disease in chickens and why is it important in swine raising? Page 258.

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Coming Meetings

Notices of coming meetings must be received 30 days before date of publication.

American Veterinary Medical Association. Ninety-seventh annual meeting. Denver-Hilton Hotel, Denver, Colo., Aug. 15-18, 1960. H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

American Humane Association. Annual convention. La-Salle Hotel, Chicago, Ill., Sept. 26-28, 1960. Mr. R. T. Phillips, 896 Pennsylvania St., Denver 3, Colo., executive director.

National Association of Artificial Breeders. Thirteenth annual convention. Brown Hotel, Louisville, Ky., Aug. 21-24, 1960. Dr. H. A. Herman, 10 North Ninth St., Columbia, Mo., executive secretary.

Central Indiana Veterinary Medical Association. Annual small animal seminar. Claypool Hotel, Indianapolis, Ind., Sept. 14, 1960. P. T. Parker, 5901 Crawfordsville Rd., Speedway 24, Ind., chairman.

New York State Veterinary Medical Society. Sixty-ninth annual meeting. Hotel Syracuse, Syracuse, Sept. 14-16, 1960. Joan S. Halat, New York State Veterinary Medical Society, 803 Varick Street, Utica, N. Y., assistant executive secretary.

Ninth U. S. Civil Defense Council Conference: Medical Health Section. Leamington Hotel, Minneapolis, Minn., Sept. 21-22, 1960. Dr. Carroll P. Hungate, 535 Argyle Building, Kansas City 6, Mo., chairman.

West Virginia Veterinary Medical Association. Annual meeting. Shenandoah Hotel, Martinsburg, West Va., Sept. 25-26, 1960. Dr. Harry J. Fallon, 200 Fifth St. West, Huntington, West Va., secretary.

American Veterinary Radiology Society. Annual Film Reading Session with speakers, 1 p.m. E.S.T., Sept. 25, 1960. Hotel Elkart, Elkhart, Ind. J. J. Fishler, Elkhart, Ind., secretary.

Armed Forces Institute of Pathology. Seventh annual course. Armed Forces Institute of Pathology, Washington, D.C., Sept. 26-30, 1960. Deadline for applications is August 15. To apply, write: The Director, Armed Forces Institute of Pathology. Washington 25, D.C.

Illinois Conference and Extension Short Course for Veterinarians. College of Veterinary Medicine, University of Illinois, Urbana, Oct. 6-7, 1960. Dr. L. E. Boley, Chairman 1960 Veterinary Conference.

Helminthological Society of Washington. Fiftieth anniversary. Scientific program will be conducted at the University of Maryland, College Park, Md., Oct. 8, 1960. Helminthological Society of Washington, Animal Disease and Parasite Research Branch, ARS, USDA, Beltsville, Md., publicity committee.

Gaines Dog Research Center. Tenth annual symposium. Kankakee Civic Auditorium, Kankakee, Ill., Oct. 12, 1960. Dean C. A. Brandly, School of Veterinary Medicine, University of Illinois, Urbana, Ill., chairman.

Purdue University. Forty-eighth annual conference for veterinarians. School of Veterinary Science and Medicine, Purdue University, Lafayette, Ind., Oct. 12-14, 1960. Erskine V. Morse, dean.

Eastern Iowa Veterinary Association, Inc. Forty-seventh annual meeting. Hotel Montrose, Cedar Rapids, Oct. 13-14, 1960. Charles B. Thayer, Medical Laboratory Center, S. U. I., Iowa City, Iowa, secretary.

Continued on adv. p. 46

Proud owner Barbara Clausen is shown beside Black Orbit, 17-month-old Angus steer judged Grand Champion Steer at 1959 Illinois State Fair.

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COMING MEETINGS—continued from adv. p. 44

New England Veterinary Medical Association. Twenty-sixth annual meeting. Sheraton Biltmore Hotel, Providence, R.I., Oct. 16-19, 1960. L. T. Maloney, New England V.M.A. Consultant, 6 Beacon St., Boston, Mass.

United States Livestock Sanitary Association. Sixty-fourth annual meeting. Daniel Boone Hotel, Charleston, W. Va., Oct. 19-21, 1960. R. A. Hendershott, 33 Oak Lane, Trenton 8, N.J., secretary.

Southern Veterinary Medical Association, Inc. Annual meeting. Francis Marion Hotel, Charleston, S.C., Oct. 23-26, 1960. Otto M. Strock, 461 Maybank Highway, Charleston, S.C., general chairman.

Animal Care Panel. Annual convention. Sheraton-Jefferson Hotel, St. Louis, Mo., Oct. 26-28, 1960. Herbert Graff, 835 S. 8th St., St. Louis, Mo., convention secretary.

Cornell University. Annual nutrition conference for feed manufacturers. Statler Hilton Hotel, Buffalo, N.Y., Nov. 2-4, 1960. For programs, preregistration and hotel reservation cards, contact: Prof. Harold H. Williams, Savage Hall, Cornell University, Ithaca, N.Y., chairman.

Missouri, University of. Thirty-sixth annual veterinary conference. University of Missouri, School of Veterinary Medicine, Columbia, Mo., Nov. 7-8, 1960. Cecil Elder, School of Veterinary Medicine, Veterinary Pathology, University of Missouri, chairman.

Arizona Veterinary Medical Association. Annual meeting. Safari Hotel, Scottsdale, Ariz., Nov. 13-15, 1960. Elmer B. Powell, 1102 S. Scottsdale Rd., Scottsdale, Ariz., local arrangement (phone—WH 5-6479).

Veterinary-Nutrition Conference. Sponsored by Midwest Feed Manufacturers Association and Iowa, Kansas, Missouri, Oklahoma, and Nebraska Veterinary Medical Associations. Continental Hotel, Kansas City, Mo., Dec. 12-13, 1960. Dr. James Bailey, Walnut Grove Products Co., Atlantic, Iowa, chairman.

Wisconsin Veterinary Medical Association. Forty-fifth annual meeting. Schroeder Hotel, Milwaukee, Wis., Jan. 15-17, 1961. W. J. O'Rourke, 540 W. Washington Ave., Madison 3, Wis., secretary.

Arkansas Veterinary Medical Association. Annual meeting. Hotel Marion, Little Rock, Jan. 22-24, 1961. Thayer D. Hendrickson, 7824 Cantrell Rd., Little Rock, Ark., secretary-treasurer.

Continued on adv. p. 48.

BREAK THROUGH THE EARWAX BARRIER WITH SEBUMSOL™

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Only 1 dropperful of a safe, new drug now offers greater success in ear diagnosis and therapy. Called 'SEBUMSOL', it provides deep penetration of waxy deposits and thoroughly cleans the ear canal without irritation.

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(*3*) **Cleans the ears; facilitates aural examination and diagnosis.**

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COMING MEETINGS—continued from adv. p. 46

Minnesota Veterinary Medical Association. Annual meeting. Leamington Hotel, Minneapolis, Minn., Jan. 23-25, 1961. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, Minn., secretary.

Ohio State Veterinary Medical Association. Annual meeting. Commodore Perry Hotel, Toledo, Ohio, Feb. 5-8, 1961. Dr. R. E. Rebrassier, 1411 West Third Ave., Columbus 12, Ohio, executive secretary.

Foreign Meetings

Second International Course on Lyophilization. Lyon, France, Aug. 29—Sept. 9, 1960. For full details, contact: Dr. Louis R. Rey, Directeur des Cours Internationaux de Lyophilisation, Laboratoire de Physiologie, Ecole Normale Superieure 24, rue Lhomond, Paris 5, France.

Fourth International Congress on Animal Reproduction. The Hague, Netherlands, June 5-9, 1961. For additional information contact: the Secretariat of the Fourth International Congress on Animal Reproduction, 14, Burgemeester de Monchyplein, The Hague, Netherlands, Dr. L. Hoedemaker, secretary to the organizing committee.

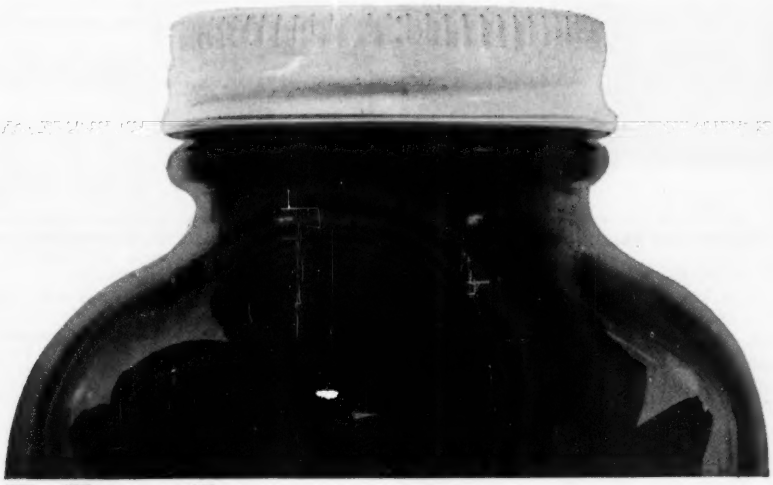
AVMA Research Fellowships Available

The Council on Research of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1961-1962.

The recipient of a fellowship must be a veterinarian and a citizen of the United States or Canada. Veterinary students who expect to graduate at the end of the current school year and who wish to follow a career in research may apply for a fellowship.

It is advisable that completed application forms be filed by Jan. 1, 1961, to allow time for correcting omissions and for some reasonable delay in arrival of letters of information from third parties. The latest date for filing the completed application is Feb. 1, 1961. Approximately one month is required for processing completed applications after receipt by the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Council on Research will meet early in March to consider applications and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from 100 per month upward. Any qualified person interested in graduate training may obtain application blanks and other information from the deans of the various colleges of veterinary medicine or by writing to the Council on Research, American Veterinary Medical Association, 600 S. Michigan Ave., Chicago 5, Ill.



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Medical organizations or individuals will no longer have to wait until they amass the cash to purchase the latest and best equipment to help their patients. Private groups and individuals can have the benefits of using the equipment immediately, preserve their liquid funds, and charge the leasing costs as operating expenses.

The lease plan will be conducted through the facilities of the Standard X-Ray Company and the Nationwide Leasing Company and will be available nationally from all Standard X-Ray Company sales representatives. Further information may be obtained by writing Nationwide Leasing Company, 11 S. LaSalle St., Chicago 3, Ill.

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Wanted—Veterinarians

Wanted—associate veterinarian for mixed practice in Central Oklahoma. State qualifications and salary requirements. Address Box G 56, JOURNAL of the AVMA.

Wanted—veterinarian to work with artificial breeding business, general practice in large purebred holstein herd and cattle sales business. Background in biochemistry or animal pathology preferred. Located near Watertown, Wis. Contact Piper Bros. Farms, Watertown, Wis.

Associate veterinarian wanted in active large animal practice in Alberta, Canada. Opportunity to take over practice. Some real estate. Give age, previous engagements and initial salary expected. No provincial board examinations required in Alberta for graduates from accredited colleges. Address Box H 54, JOURNAL of the AVMA.

Progressive, well-equipped modern small animal hospital, AAHA, has position open for a veterinarian capable of doing good-quality surgery. Job is approximately 75% surgery and 25% clinic. Chicago suburb. Address Box H 58, JOURNAL of the AVMA.

STATE OF CALIFORNIA EXECUTIVE POSITION

California needs a Chief, Public Health Laboratory Veterinary Services, to plan, organize and direct the administration of Animal Care Laws and the veterinary laboratory. Salary range: $710 to $862. Must have two years of responsible administrative experience in animal laboratory or related activity, and be able to obtain a California license within 12 months. Write at once to the **STATE PERSONNEL BOARD, 801 Capitol Ave., Sacramento 14, Calif.**

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Wanted—veterinarian with Missouri license to assist in small animal practice. Modern facilities. Salary plus. First year minimum guarantee—$8,000. Give full particulars in reply. Address Box H 57, JOURNAL of the AVMA.

Wanted—young veterinarian to assist in small animal practice. Desirable Ohio location. Address Box H 56, JOURNAL of the AVMA.

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Veterinarian seeks position with small animal practitioner in Virginia. Licensed, experienced, available immediately. Address Box H 4, JOURNAL of the AVMA.

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Graduate (MSU '58), married, completing military obligation late October, 1960, desires position in small animal practice. Experienced in hospital management and personal relations. Licensed in Michigan, Illinois. Prefer Midwest location. Address Box H 63, JOURNAL of the AVMA.

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Veterinarian, age 31, married, small animal experience, desires research position with institution or commercial organization. Please give complete information in first letter. Address Box H 53, JOURNAL of the AVMA.

Minnesota graduate desires small animal hospital position in Minnesota, Wisconsin, Florida, Michigan. Age 27, available in October. Married. Address Box H 64, JOURNAL of the AVMA.

Graduate (GA '58), desires position with small animal practitioner. Licensed in Georgia and Florida. Military obligation completed in September. Address Box H 65, JOURNAL of the AVMA.

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For sale—retirement and other interests are reasons for selling mixed practice, consisting of modern large and small animal hospitals. Located in Midwest. Suitable for 2 veterinarians. Sizable amount of cash required. Balance long term. Address Box H 52, JOURNAL of the AVMA.

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For sale—outstanding small animal hospital on San Francisco peninsula. Ideal 2-man practice, exclusive location, unlimited possibilities, completely equipped. Capacity—200. Substantial capital required. Address Box H 61, JOURNAL of the AVMA.

Missouri prosperous mixed practice in cornbelt area for sale. Modern small animal hospital, house, equipment, etc. included. Grossing in excess of $30,000. Priced at real estate value. Low down payment. Address Box H 59, JOURNAL of the AVMA.

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For sale—general practice, house and office in central Minnesota. 1959 gross over $30,000. Unlimited possibilities for 1 or 2 veterinarians. Address Box H 51, JOURNAL of the AVMA.

For sale—established Central Illinois general practice. Residence, office, equipment, drugs and office furniture. Easy terms. Address Box F 2, JOURNAL of the AVMA.

For sale or lease—modern small animal hospital in progressive Ohio suburban community. Gross $50,000 in fifth year. Excellent clientele. Located on main highway. Large, beautifully landscaped lot. House on adjacent lot optional. Address Box H 66, JOURNAL of the AVMA.

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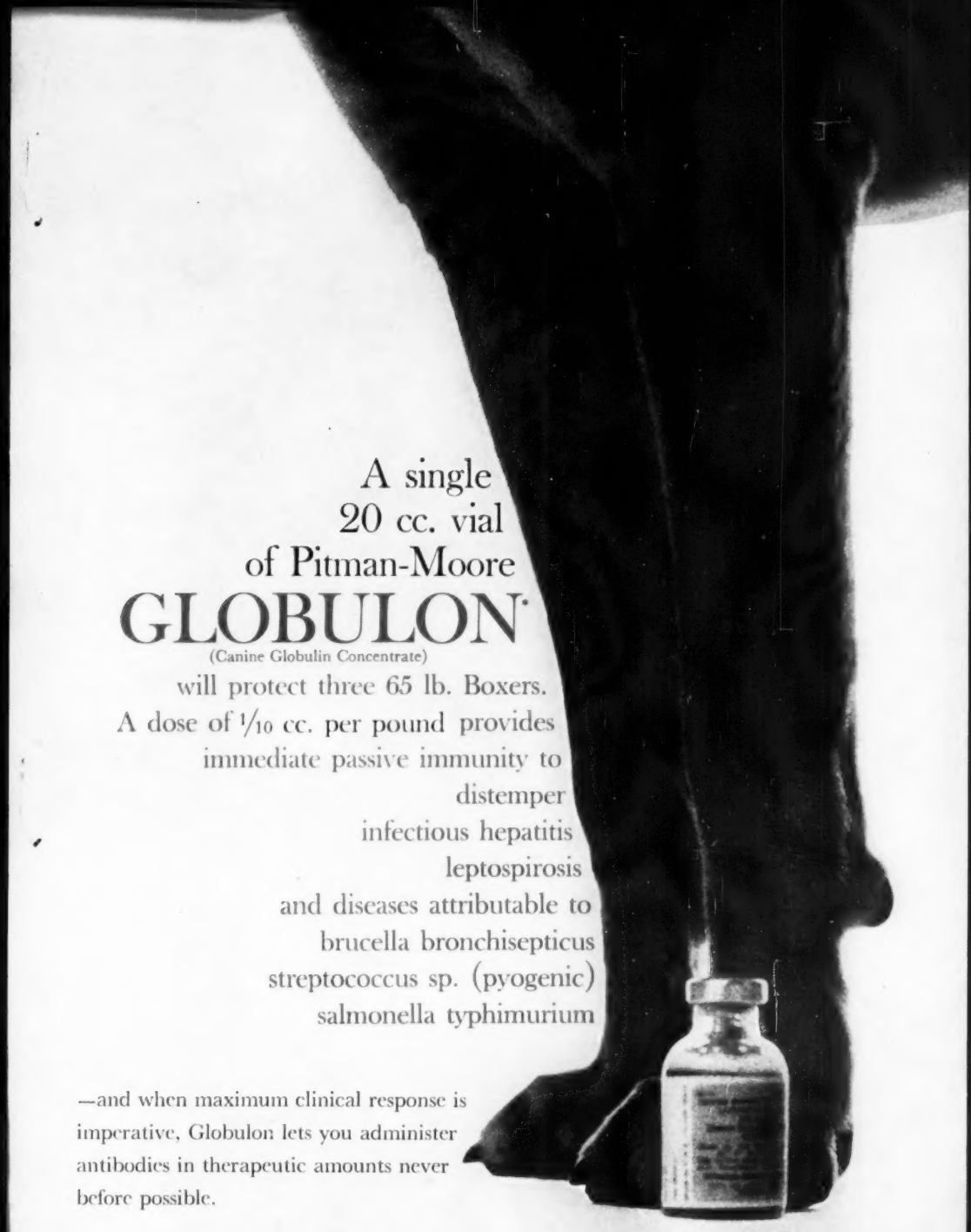

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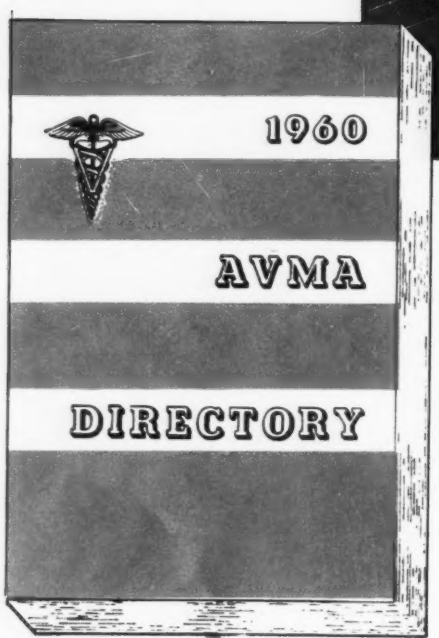

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